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THE EFFECTS OF PAIR-WEIGHING AND EXERCISE UPON THE STORAGE AND UTILIZATION OF ENERGY SOURCES

by

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Abstract

Sixty-two male Wistar rats were randomly assigned to a pair-weighed and an ad libitum feeding group with half of the animals in each group exercised five days per week, for one hour per day at 1.0 m.p.h. for 10 weeks. Measurements of liver and muscle glycogen, plasma and epididymal fat pad FFA release and blood glucose levels were taken. All sedentary animals were sacrificed at rest, while some trained animals were sacrificed at rest and others after an exhaustive treadmill run. Pair-weighing had no significant effects on plasma or tissue metabolites under any exercise condition. Training resulted in increased adrenal weights and a decrease in epididymal fat pad weights. Exhaustive exercise produced a significant decrease in liver glycogen content with blood glucose levels, with an increase in plasma and adipose tissue FFA levels and plasma lactate concentrations. It was concluded that with a moderate treadmill training program the dietary restriction induced by pair-weighing did not effect the storage and utilization of energy sources.

Dedication

To my family, with love and gratitude:

so far away in miles, yet always close

with words of love and encouragement

throughout all my endeavours.

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CHAPTER I

INTRODUCTION

Introduction

Numerous investigators have shown that young animals engaged in a regular training program gain weight more slowly than sedentary controls (Steinhaus et al, 1932; Kimeldorf and Baum, 1954; Dalhgren, 1965; Bloor et al, 1968; Crews et al, 1969; Buuck and Tharp, 1971). The significantly lower final body weights of the endurance trained animals has been attributed to several factors: (1) the increased caloric expenditure; and (2) the depressant effect of exercise on appetite (Crews et al, 1969; Oscai et al, 1971a; Oscai et al, 1972). These body weight differences have been related to total body composition, with exercised animals having a reduced total-body fat content (Oscai et al, 1972).

Recently, in an attempt to minimize these differences, some investigators have employed a specific program of dietary restriction (pair-weighing) to maintain equal body weights between exercised and sedentary animals. Both Oscai and Holloszy (1969) and Crews et al (1969) studied the effects of exercise and caloric restriction in exercised, pair-weighed sedentary and "free-eating" sedentary animals and found a decrease in lean body mass and a reduced proportion of fat in both exercised and pair-weighed animals, though the decrease in fat content was not as great in the pair-weighed rats as the exercised rats. Oscai et al (1972) also confirmed the above findings and found that training and caloric restriction at a young age reduced the size and number of epididymal fat cells in rats.

Statement of the Problem

The main purpose of this study was to determine if the technique of pair-weighing exercised and sedentary animals to maintain equal body weights had any effect on the storage and utilization of energy sources. The following parameters were measured: total body weight, organ weights, liver and skeletal muscle glycogen, blood and gastrocnemius muscle lactate, blood glucose, plasma free fatty acid (FFA) levels and lipid FFA mobilization.

Rationale Behind the Study

From the foregoing discussion it would appear that there are significant differences between ad libitum fed sedentary, pair-weighed sedentary and exercised rats. However, these differences primarily demonstrate a different relationship in lean body mass. The ad lib sedentary animals have more adipose tissue than either the sedentary pair-weighed or ad lib exercised animals.

In view of the relationship between exercise and lipolysis, the difference in total available adipose tissue may conceivably influence the availability of fat as a substrate for energy. Indirectly, in light of the complex and interrelated metabolic pathways involved in energy release, it was decided also to study some of these related pathways, particularly the carbohydrate anaerobic metabolic paths. In addition, selected organs used in energy storage or release were examined.

Limitations and Delimitations of the Study

1. The study was confined to male Wistar rats between the ages of 6 to 22 weeks.

2. Only one intensity of endurance training was used, and some trauma could have resulted from the presence of the electrical shocking device on the treadmill.

3. Body composition measurements were not obtained.

4. Differences in body weights were not accounted for when analyzing differences in organ weights.

CHAPTER II

REVIEW OF THE LITERATURE

TOTAL BODY AND ORGAN WEIGHTS

Numerous investigators have attempted to determine the influence of chronic exercise on body and organ weights (Hatai, 1915; Donaldson and Meeser, 1932, 1933; Kimeldorf and Baum, 1954; Asahino et al, 1959; Dahlgren, 1965; Bloor et al, 1968). Crews and co-workers (1969) studied young rats engaged in a moderate training program over a three month period. They found that exercising animals gained significantly less weight than sedentary controls. Similar results of a slower growth rate of exercising rats have been reported by other investigators (Steinhaus et al, 1932; Kimeldorf and Baum, 1954; Dahlgren, 1965; Bloor et al, 1968; Buuck and Tharp, 1971). The lower final body weights and slower growth rates have been attributed to the observation that exercising animals do not compensate for the increased caloric expenditure by increasing food intake (Crews et al, 1969; Oscai et al, 1971; Oscai et al, 1972). Mayer and associates (1954) noted a small decrease in food intake with exercise periods of one hour or less, with an increase in food consumption in proportion to the increase in exercise time up to six hours. Using similar mild exercise programs, Thomas and Miller (1958) and Stevenson et al (1966) both observed a decrease in food intake and body weight on exercise days with food consumption returning to normal values on

days free of exercise.

In studies using pair-weighed sedentary animals the changes in total body weight paralleled those of exercising animals (Jones et al, 1964; Oscai, Mole, and Holloszy, 1971; Oscai et al, 1972).

Donaldson and Meeser (1932) showed that exercised animals had heavier adrenals than controls and these results have since been confirmed by many other investigators (Kimeldorf and Baum, 1954; Jones et al, 1964; Gollnick, Struck and Bogyo, 1967; Buuck and Tharp, 1971). Bloor et al (1968), however, found no differences in adrenal weights between trained and untrained rats.

Hatai (1915) reported heavier liver, kidney and testicular weights for exercised animals, however, conflicting observations were made by Jones et al (1964) for liver weights. Also, decreased kidney weights were noted in trained rats (Kimeldorf and Baum, 1954; Gollnick and Hearn, 1961; Bloor et al, 1968). An unexplained decrease in testicular weights with exercise has been reported by Tipton, Terjung and Barnard (1968) and Steinhaus et al (1932).

The effects of exercise on spleen weights appear to be dependent upon the exercise state of the animal at sacrifice. Tipton, Tharp and Schild (1966) and others have noted a decrease in spleen weight with fatigue (Barcroft and Florey, 1929-30; Gollnick et al, 1965; Gollnick, Struck and Bogyo, 1967). At rest, no difference between exercise and control groups for spleen weight has been reported (Tipton, Tharp and Schild, 1966; Bloor et al, 1968).

Cardiac hypertrophy has been a characteristic physiological adaptation to endurance training in rats (Hatai, 1915; Donaldson and Meeser, 1932; Kimeldorf and Baum, 1954; Van Liere et al, 1965; Van Liere and Northup, 1967). Oscai et al (1971a) found with a strenuous treadmill training program that trained animals had significantly lighter heart weights than sedentary "free-eating" controls. In addition, pair-weighed sedentary animals had hearts weighing significantly less than either group. The heart weight to body weight ratio in Oscai's study, however, showed exercised rats to have significantly heavier hearts than either sedentary group. Employing swimming programs of various intensities, Oscai et al (1971a) observed a cardiac hypertrophy only in groups exercised 60 and 180 min/day. In a similar swimming study using animals more strenuously exercised (6 hours of daily swimming), Oscai et al, (1971b) found trained rats had lighter hearts than sedentary "free-eaters" though it was not statistically significant. However, pair-weighed sedentary rats had smaller heart weights than either exercise animals or sedentary "free-eaters".

SKELETAL MUSCLE AND LIVER GLYCOGEN AND BLOOD GLUCOSE

Glycogen is a complex moiety by which carbohydrate is stored in both liver and muscle. Liver glycogen can be hydrolyzed to form glucose which is then released to maintain normal blood glucose levels, while muscle glycogen is degraded and its product utilized during periods of increased energy demand.

Glucose derived from the blood and skeletal muscle glycogen are phosphorylated to glucose-6-phosphate by the action of the enzymes hexokinase and phosphorylase, respectively. At this point, glucose-6-phosphate can be utilized in the muscle for the anaerobic production of energy via the glycolytic pathway. (Harper, 1971).

Normal resting values for skeletal muscle glycogen in humans have been reported to be in the order of 1.5 - 2.0 gm/100 gm of tissue (Ahlborg et al, 1967; Bergstrom et al, 1967; Hermansen et al, 1967; Costill et al, 1971). Resting muscle glycogen in albino rats has been shown to be slightly lower: Fielding (1973) and Gollnick and King (1969) have observed glycogen concentrations in the range of 2.0 - 3.2 mg/gm in the biceps brachii muscle and Fielding (1973) has noted a glycogen content of 3.5 mg/gm in resting gastrocnemius muscle.

Although training has been shown to cause increased skeletal muscle glycogen stores in dogs, (Proctor and Best, 1932) rats (Gollnick and King, 1969) and in humans (Taylor et al, 1971) other investigators have not found increases in either skeletal muscle glycogen in humans (Hermansen et al, 1967) or rats (Gollnick et al, 1970; Fielding, 1973) or in liver glycogen in rats (Gollnick and King, 1969; Gollnick et al, 1970).

Skeletal muscle and liver glycogen stores have been noted to decrease (Costill et al, 1971) or to be almost depleted (Ahlborg et al, 1967; Gollnick and King, 1969; Gollnick et al, 1970; Karlsson and Saltin, 1971) as a result of heavy prolonged work to exhaustion.

In heavy intermittent exercise, Hermansen et al (1967) noted glycogen depletion with the greatest reduction in glycogen content occurring during the first 20 min work period. Bergstrom et al (1971) and Taylor et al (1971) have also observed this rapid glycogenolysis during the early phases of the exercise bout. Both Bergstrom et al (1967) and Ahlborg et al (1967) observed a correlation between performance time and the initial muscle glycogen content.

On the other hand, short-term high intensity exercise to exhaustion has been reported to result in either a slight decrease (Costill, 1971) or no change (Taylor et al, 1971) in muscle glycogen levels.

The normal resting concentration of plasma glucose is maintained fairly constant via hormonal regulation at approximately 90 - 100 mg% in humans (Mountcastle, 1968). Resting values for plasma glucose for laboratory albino rats have been reported in the range of 115 - 120 mg% (Fielding, 1973; Taylor and Rao, 1973). Investigators studying the effects of severe submaximal exercise have generally noted no differences in resting plasma glucose levels between trained and untrained rats (Fielding, 1973; Taylor and Rao, 1973) or humans (Keul, Doll and Keppler, 1972); however, lower glucose levels have been found in trained athletes versus untrained subjects at rest (Johnson et al, 1969).

Prolonged moderately heavy work to exhaustion produces a significant decrease in blood glucose levels (Bergstrom et al, 1967;

Gollnick et al, 1970; Keul, Doll and Keppler, 1972; Taylor and Rao, 1973). Both Ahlborg et al (1967) and Taylor et al (1971) observed an initial increase in blood glucose levels followed by a decline to exhaustion.

In prolonged mild exercise Young et al (1966, 1967) noted a gradual decline in blood glucose, which stabilized at a slightly lower level after 9 hours of exercise. They also observed an increased synthesis of glucose during exercise. In exercise of a shorter duration Reichard et al (1961) also found a decrease in blood glucose.

In intermittent maximal runs on a treadmill Hermansen et al (1970) found an increase in blood glucose levels from resting values of 82.6 mg% to 170.7 mg%. Pruett (1970) showed that near maximal efforts requiring 85% of maximal aerobic capacity resulted in a decrease in blood glucose during the first 10 minutes of the work bout followed by a hyperglycemia in exercise lasting longer than 10 minutes. She attributed the increased hepatic glucose output to a rise in circulating catecholamines.

BLOOD AND MUSCLE LACTATE

Lactate is the end-product of anaerobic metabolism. Under aerobic conditions pyruvate is the normal end metabolite of glycolysis; however, under conditions of anaerobiosis pyruvate is reduced to lactate by NADH (nicotinamide-adenine dinucleotide), with the reaction catalyzed by the enzyme LDH (lactic dehydrogenase)

(Harper, 1971). Lactate is known to diffuse into the blood and, prior to the development of the biopsy techniques for the assay of tissue metabolites, plasma lactate was considered to be the prime indicator of the involvement of anaerobic processes. Removal of lactate from the blood has been noted in the heart (Carlsten et al, 1961), and liver (Rowell et al, 1965, 1966, 1968). The fate of lactate in the liver is belived to be gluconeogenesis via the Cori cycle (Harper, 1971), while in the heart (Gollnick, Struck and Bogyo, 1967) and in skeletal muscle (Stainsby and Welch, 1966) lactate may be used as a substrate. Normal resting levels of plasma lactate in humans is approximately 10 mg% (Mountcastle, 1968). Taylor and Rao (1973) and Fielding (1973) have reported comparable resting plasma and muscle lactate in rats ranging from 15.0 - 25.0 mg%.

There is a concensus in the literature that trained subjects accumulate lower blood lactate concentrations than untrained subjects as a result of exercise at the same relative submaximal intensity (Astrand, 1956; Hermansen et al, 1967; Williams et al, 1967). Following short-term maximal exercise however, trained subjects have been reported to elicit a higher (Cunningham and Faulkner, 1969; Ekblom et al, 1968) or a lower (Johnson and Brouha, 1942; Holmgren and Strom, 1959) blood lactate concentration using an absolute and a relative work load, respectively. During exercise periods of a given duration, plasma lactate levels have been shown to be positively related to the relative intensity of the

exercise. This relationship has been illustrated by Knuttgen (1962) using 20 minute exercise bouts of different intensities, and by Knuttgen and Saltin (1972) using 4 minute exercise bouts. This rise in blood lactate concentration is attributed to the enhanced involvement of anaeroboc metabolism as intensity of exercise increases (Knuttgen, 1962; Nagle et al, 1970), and is more pronounced at intensities above 60% of $^{V}O_2$ max (Knuttgen and Saltin, 1972).

At a given submaximal intensity of exercise, blood lactate levels tend to decrease as the duration of the exercise is extended. Investigators have noted that, at intensities from approximately 60 to 80 % $^{V}O_2$ max, blood lactate concentration rose during the first minutes of exercise and subsequently tended towards resting values as the duration of the exercise was prolonged in men (Ahlborg et al, 1967; Bergstrom et al, 1967; Saika et al, 1967) and rats (Dawson et al, 1971). In addition, even when the prolonged submaximal work has been extended to exhaustion in marathon runners (Costill, 1970), in cross-country skiers (Astrand et al, 1963) and in rats (Taylor and Rao, 1973; Fielding,1973) blood lactate levels approaching resting values have been observed.

With maximal exercise there is general agreement that lactate is continuously produced throughout the exercise period (Klausen, Knuttgen and Forster, 1972; Keul and Doll, 1973; Hermansen et al, 1970), primarilv due to the predominance of anaerobic metabolism in this type of exercise (Astrand and Rodahl, 1970).

PLASMA AND ADIPOSE TISSUE FREE FATTY ACIDS

Fat, stored in the form of triglycerides in adipose tissue, is known to be an energy depot which can be called upon in times of increased caloric requirements for purposes of energy production. In adipose tissue the conversion of triglycerides into glycerol and FFA is catalyzed by a hormone-sensitive lipase. Free fatty acids, which can be readily oxidized by muscle tissue, can then be transported via the circulatory system to the site of utilization. The significance of the production of FFA in adipose tissue, its subsequent release into the blood and its utilization during exercise has been emphasized in recent research (Carlson et al, 1963; Havel et al, 1963; Paul and Issekutz, 1967).

During rest in humans, plasma FFA concentrations range from 0.36 to 0.80 uEq/ml (Havel and Goldfien, 1959; Basu et al, 1960; Rodahl et al, 1964; Young et al, 1966; Johnson et al, 1969; Taylor et al, 1971). In laboratory rats, resting plasma FFA levels have been reported at approximately 0.3 uEq/ml (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1972; Fielding, 1973). The concentration of FFA in adipose tissue (epididymal fat pads) of resting normal rats is approximately 2.0 - 3.0 uEq/gm of tissue (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1972; Fielding, 1973). Exercise training has no effect on resting plasma FFA levels in humans (Johnson et al, 1969), dogs (Issekutz et al, 1965; Paul and Issekutz, 1967) and rats (Gollnick, 1967; Gollnick et al, 1970;

Taylor, 1972; Fielding, 1973). As well, adipose tissue FFA concentrations of trained and untrained rats have been found to be similar (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1972; Fielding, 1973).

Plasma FFA concentrations have been observed to increase with increasing intensity of exercise (Keul et al, 1967; Taylor et al, 1971). Keul et al (1967) using continuous bicycle exercise during which the load was increased by 50 Watts every six minutes found a concomittant rise in arterial FFA levels and arteriovenous differences. Similarly, Taylor et al (1971) noted a progressive rise in plasma FFA concentrations using graded relative work loads, and attributed this primarily to an increased lipolysis of adipose tissue triglycerides.

Submaximal exertion elicits a gradual rise in plasma FFA levels as work is continued (Carlson and Pernow, 1961; Havel et al, 1963; Friedberg et al, 1963; Rodahl et al, 1964; Issekutz et al, 1965; Gollnick, 1967; Keul et al, 1967; Johnson et al, 1969; Taylor et al, 1971). This rise has been attributed to an increased lipolysis based upon: (1) an increased activity of the sympathetic nervous system (Havel and Goldfien, 1959; Havel et al, 1963; Carlson et al, 1963; Havel et al, 1964; Carlson et al, 1965; Gollnick, 1967), (2) an increased level of circulating hormones (Carlson and Pernow, 1959; Havel and Goldfien, 1959; Schotz and Page, 1959; Vendsalu, 1960; Basu et al, 1960; Friedberg et al, 1963; Havel et al, 1963; Carlson et al, 1963; Miller et al, 1963; Rodahl

et al, 1964; Parizkova and Stankova, 1964; Gollnick, 1967; Taylor et al, 1971), and (3) the lower lactate concentration of the blood with prolonged submaximal work (Issekutz et al, 1965, 1966; Johnson et al, 1969; Taylor et al, 1971). This characteristic rise in plasma FFA occurs even though evidence suggests an increased utilization of the plasma lipids by working muscles during prolonged exercise (Carlson and Pernow, 1959; Friedberg et al, 1960; Carlson and Pernow, 1961; Miller et al, 1963; Friedberg et al, 1963; Havel et al, 1964; Issekutz et al, 1966; Jones and Havel, 1967; Havel et al, 1967; Paul and Issekutz, 1967; Keul et al, 1967).

Adipose tissue FFA levels have been noted to follow a similar pattern in rats during extended submaximal exercise (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1973; Fielding, 1973).

During high intensity exercise of short duration, plasma FFA concentrations are either unchanged from resting values (Carlson and Pernow, 1959; Keul et al, 1972) or decrease slightly (Carlson and Pernow, 1959; Rodahl et al, 1964). This is expected in view of the high proportion of anaerobic metabolism in this type of work (Astrand and Rodahl, 1970) and the high production of lactate (Hermansen, 1969) which has been noted to inhibit FFA mobilization and oxidation (Gold et al, 1963; Issekutz et al, 1965).

Trained subjects have been observed to have a lower (Johnson et al, 1969) or a higher (Cobb and Johnson, 1963; Issekutz et al, 1965; Keul et al, 1972) plasma FFA concentration than

untrained subjects during extended submaximal exercise. Johnson et al (1969) attributed the lower values in athletes to an enhanced utilization of the substrate, whereas the higher values for trained subjects have been primarily explained to be due to a higher lipolytic rate (Gollnick and Williams, 1969) or a decreased proportion of glycolysis (Issekutz et al, 1965; Keul et al, 1972).

CHAPTER III

METHODS AND PROCEDURES

Sixty-two male Wistar rats, 6 - 8 weeks of age, with initial body weights ranging from 180 grams to 230 grams were used in this study. The animals were housed in individual 7 X 10 X 7 inch suspended cages in a temperature controlled room maintained at 24° C ($\pm$ 1°C) with a 12 hour light-dark cycle.

Following the initial training period described below, 30 animals were weight matched and assigned to the pair-weighed feeding group and 32 animals were placed in an ad libitum feeding group. Fifteen animals in the pair-weighed group and 19 animals from the ad libitum group served as non-exercise controls while the remaining animals were trained to run on a motor driven treadmill (Quinton Rodent Treadmill or Collins Treadmill). The training regime consisted of running at progressively increasing speeds until, at the end of three weeks, each animal was able to run for one hour at one mile per hour, five successive days per week. The animals were maintained on this program for a further 10 - 14 weeks. Mild electrical stimulation (140 mv maximum) was used as an initial training stimulus to train the rats to run.

All animals were fed standard rat chow (Rockland Complete Rat Diet) and fresh water was available at all times. The animals in the pair-weighed group were weighed daily on a Triple Beam Balance prior to the exercise period; the weights recorded and the food intake adjusted daily to maintain approximately equal body weights ($\pm$ 10 grams) between

the exercise animal and its matched control. This maintenance of equal body weights required that the quantity of the food intake of the sedentary pair-weighed animals be severely restricted. The animals in the ad libitum group were weighed once a week and the weights were recorded. These rats had access to food and water ad libitum.

Five animals from the pair-weighed group, eight animals from the ad libitum group, and all control animals were sacrificed at rest. The remaining 10 animals in the pair-weighed group and 5 animals from the ad libitum group were sacrificed after an exhaustive run on the treadmill. All animals were weighed prior to sacrifice on a Triple Beam Balance. Those exercised to exhaustion were weighed prior to the run since animals were found to lose 5-10 grams after a normal training run.

On the morning of sacrifice, the animals were anaesthetized with ether and an incision made along the abdominal midline. The abdominal aorta was isolated, a 20 guage heparinized syringe inserted at the aortic bifurcation and 8-11 cc of blood withdrawn. The blood was transferred to a centrifuge tube, spun down at 1900 rpm in an International Refrigerated Centrifuge Model PR-6, and the plasma removed, frozen immediately in a dry ice and alcohol solution and stored in a deep freeze (50°K) for later analysis of free fatty acid (FFA), glucose and lactate content. After exsanguination, a thoracotomy was performed, the liver removed immediately and weighed on a Triple Beam Balance. A small sample ($>$ 300 mg) was removed, weighed and placed in a screw-cap test tube and frozen as described for glycogen analysis at a later date.

The heart, spleen, adrenals, right kidney and testes were removed, cleared of excess fatty tissue, and weighed on a Roller-Smith Precision Balance. The epididymal fat pads were removed, weighed, placed in a screw-cap tube, and frozen for later FFA analysis. The left biceps brachii and both gastrocnemius muscles were removed, weighed, placed in a screw-cap tube, and frozen for analysis of muscle glycogen and lactate content. The total procedure was completed within 15 minutes.

Glycogen Determination

Liver and muscle glycogen was determined using the method of Lo, Russell, and Taylor (1970). The frozen tissue samples of liver, biceps brachii muscle and gastrocnemius muscle were placed in screw-cap tubes, completely covered (0.5 ml for biceps brachii; 1.0 ml for liver and gastrocnemius) in a solution of 30% potassium hydroxide (KOH) saturated with sodium sulfate (Na_2SO_4). The tubes were capped, placed in a boiling water bath for 20 - 30 minutes until a homogenous solution was obtained, then cooled in an ice bath.

The glycogen was precipitated from the alkaline digestate by the addition of 1.1-1.2 volumes of 95% ethanol. The samples were placed on ice for 30 minutes, centrifuged at 810 x g for 20-30 minutes, and the supernatants were aspirated. The glycogen precipitates were then dissolved in 10 ml of distilled water. From this solution, 0.1 ml for liver, 0.5 ml for gastrocnemius, and 1.0 ml for biceps brachii were pipetted into 150 X 20 mm test tubes with sample volume being brought to 1.0 ml with the addition of distilled water. One ml of 5% phenol solution was added to each test tube. Five ml of 96-98% sulfuric acid

(H_2SO_4) were added rapidly with the acid stream directed against the liquid surface to ensure thorough mixing. After standing for 10 minutes the samples were shaken and placed for 20 - 30 minutes in a water bath at 25 - 30°C before colormetric readings were taken.

All samples were prepared in duplicate to minimize errors resulting from contamination with cellulose lint. Absorbance readings were taken on a Bausch and Lomb Spectronic 20 at a wavelength of 490 mu. A standard curve was constructed using the average absorbancy readings of standard glycogen solutions subjected to the same phenol-sulfuric acid-glycogen reactions as the liver and muscle samples. The calculations were adjusted according to the dilution factor.

Blood Lactate Determination

Blood lactate was determined using the Sigma Kit method (1965). The plasma was allowed to thaw and 0.5 ml was transferred to a test tube containing 1.0 ml of cold 8% perchloric acid (PCA). The solution was shaken vigorously, allowed to stand for 5 minutes, and then centrifuged at 3000 rpm for 5 minutes to obtain a protein free filtrate. This filtrate was transferred to another test tube and recentrifuged, if necessary, to obtain a clear filtrate. In an 125 ml Erlyenmeyer flask, a reagent containing 32 ml of distilled water, 16 ml of glycine-hydrozine buffer (pH, 9.2), 0.8 ml lactic dehydrogenase suspension (LDH), and 80 mg of β-diphosphopyridine nucleotide (β-DPN) was mixed vigorously.

A series of six labeled test tubes containing known concentrations of the above described reagent and a known lactic acid working standard

solution (dilute 1.0 ml lactic acid standard (0.4 mg lactic acid/mg) to 5.0 ml with distilled water) were used to construct a standard curve. Other test tubes were set up in duplicate to analyze the unknown samples. Each sample tube contained 2.8 ml of reagent mixture and 0.2 ml of the protein free filtrate of the unknown sample. If the animal was sacrificed after an exhaustive run, each tube contained 2.9 ml of reagent mixture and 0.1 ml of the protein free filtrate because of the high lactate concentration expected. All tubes were mixed thoroughly and allowed to sit at room temperature for 45 minutes. Each tube was then read for optical density at a wavelength of 340 mu on a Bausch and Lomb Spectronic 20 using the first test tube in the standard curve to calibrate for zero optical density. The unknown values for blood lactate were then obtained from the standard curve, which was plotted from the optical density at 340 mu versus lactic acid concentration in milligrams per cent.

Muscle Lactate Determination

Muscle lactate concentration was measured according to the method of Lundholm et al. (1963). A small sample of muscle (200 - 400 mg) was homogenized (Virtis "23" Homogenizer) at medium speed for 5 minutes in 29 ml/g of cold 6% perchloric acid (PCA). The homogenate was centrifuged and one drop of methyl orange added to the decanted supernatant. The solution was neutralized to a pH of 3.5 (salmon pink) with potassium carbonate (K_2CO_3), and stood in an ice bath for 10 minutes. A reagent containing 1.35 ml of glycine-hydrozine buffer, 0.15 ml β-diphosphopyridine nucleotide (β-DPN), and 1.40 ml of distilled water

was prepared. Duplicate 0.1 ml samples of the extract from the homogenate were added to the reagent and two readings (E_1) 3 minutes apart were taken on a Unicam SP 1800 Ultraviolet Spectrophotometer with a wavelength of 340 mu and a slit width of .315. Twenty λ (0.02ml) of lactic dehydrogenase (LDH) was prepared immediately before use and added to the tubes. Upon completion of the reaction (10-20 minutes) the optical density (E_2) was read twice within a 3 minute interval. The factor for calculations was 158. Absorbance (E_2-E_1) multipled by 158 resulted in u moles of lactate per gram of tissue.

Plasma and Adipose Free Fatty Acid Determination

Plasma FFA and FFA content of the fat pads were analyzed according to the method of Dole and Meinertz (1960).

For the determination of plasma FFA, duplicate 1.0 ml samples of plasma were added to 5.0 ml of fat extraction mixture in screw-cap tubes, shaken vigorously, and allowed to stand for 5 minutes. Two milliliters of distilled water and 3.0 ml of heptane were added to the tubes. The tubes were shaken thoroughly and allowed to stand for 10 minutes. The 3.0 ml of the upper phase were removed, transferred to a 15 cc conical tube and 1.0 ml of Thymol blue working solution added just prior to titration with the base. Thymol blue rather than Nile blue was used as it produced a more distinct and point. A blank and standards were run for each set of determinations and a standard curve constructed. The plasma FFA concentration was derived from the standard curve and multiplied by three.

The epididymal fat pad FFA content was determined by homogenizing approximately 800 mg samples in 20 ml of fat extraction mixture in a Virtis "23" Homogenizer. Duplicate 5.0 ml aliquots of the homogenate were taken and with the addition of 2.0 ml of distilled water and 3.0 ml of heptane followed the procedure as outlined above. Calculations for tissue FFA concentration were done in the same manner as plasma FFA.

Blood Glucose Determination

Blood glucose was determined by the Glucostat* method (Worthington Biochemical Corporation, Freehold, New Jersey). The semi-micro adaptation which utilized a 0.1 ml aliquot of plasma was used. The absorbancy of the solutions was read on a Bausch and Lomb Spectronic 20 set at a wavelength of 420 mu. Glucose was calculated in milligrams per 100 ml of blood.

Statistical Analysis

The data was analyzed using a two-way analysis of variance. The Scheffé Multiple Comparison Test was used to determine the difference between mean scores (Edwards, 1972). Differences at the 0.05 level of confidence were considered to be significant.

CHAPTER IV

RESULTS

Neither training nor diet had a significant effect on total body weight (Figure 1)*. Although there were no statistically significant differences between groups at sacrifice, throughout the training period sedentary ad lib fed animals weighed more than the ad lib trained animals, with pair-weighed animals maintaining approximately equal body weights to the ad lib trained animals (Figure 2). Final body weights showed a trend for ad lib fed sedentary animals to weigh more than the other three groups.

Liver, heart, kidney and spleen weights for trained resting and fatigue animals were not significantly different (Figures 3 to 6). No differences for trained resting and sedentary control animals were noted. Control animals had significantly larger liver and spleen weights than fatigue animals, while fatigue animals had larger heart weights than sedentary controls. Fatigue resulted in lower left testicular weights than those of trained resting or control groups (Figures 7 and 8). The epididymal fat pads of control animals were greater than those of exercised animals (Figures 9 and 10). The fat pads of trained resting and fatigue animals were not statistically different, however, it was noted that trained resting animals tended to have larger fat pads. Resting trained animals had significantly larger adrenals than the control group

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*Tables for the Scheffé Comparison are found in Appendix C.

FIGURE I

LEGEND FOR ALL FIGURES

PW: Pair-weighed animals

AL: Ad Libitum animals

PWT: Pair-weighed trained

PWS: Pair-weighed sedentary

ALT: Ad Lib trained

ALS: Ad Lib sedentary

FIGURE 2

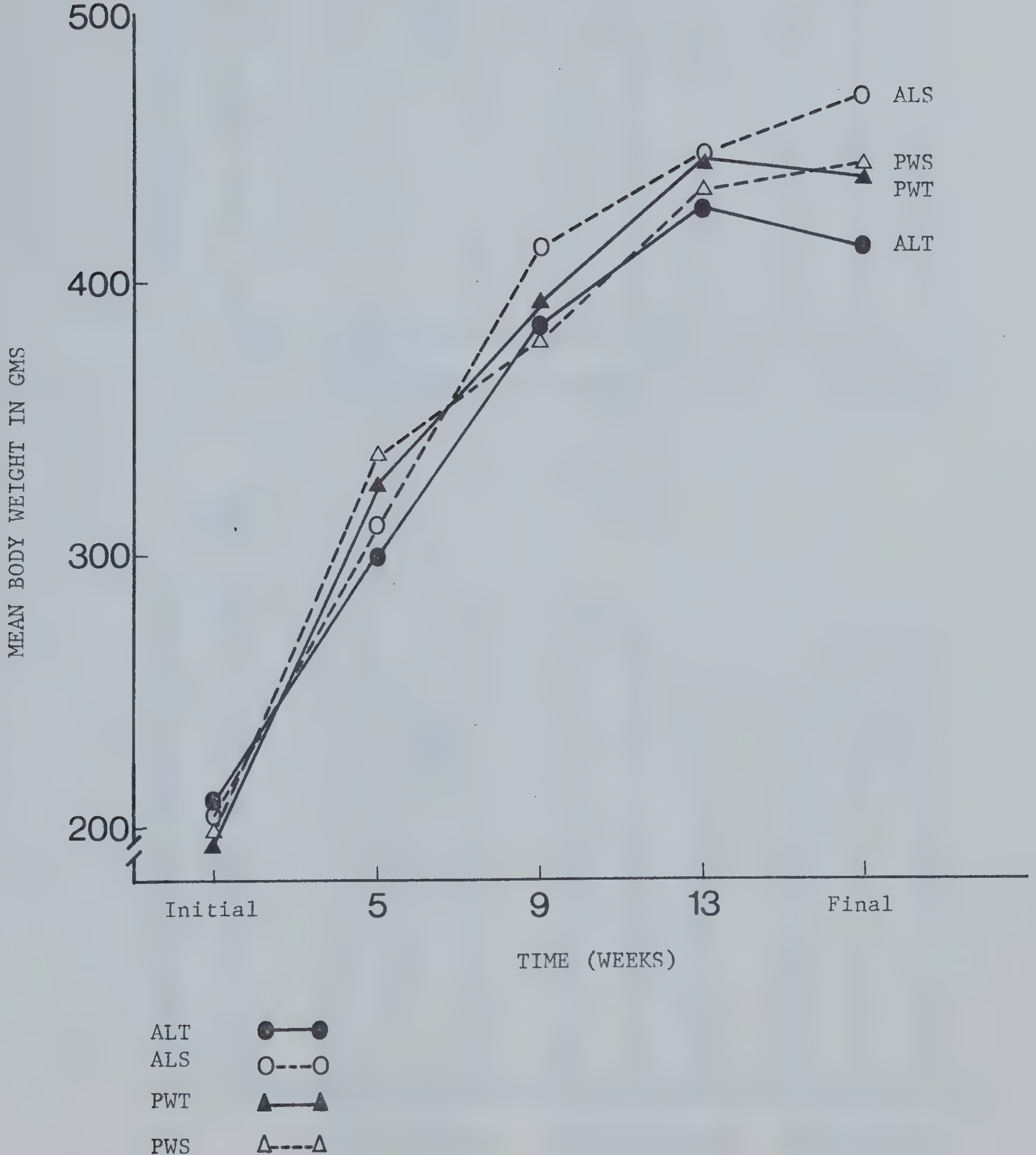

500
400
300
200
MEAN BODY WEIGHT IN GMS
Initial
5
9
13
Final
TIME (WEEKS)
ALS
PWS
PWT
ALT
ALT
ALS
PWT
PWS

FIGURE 3

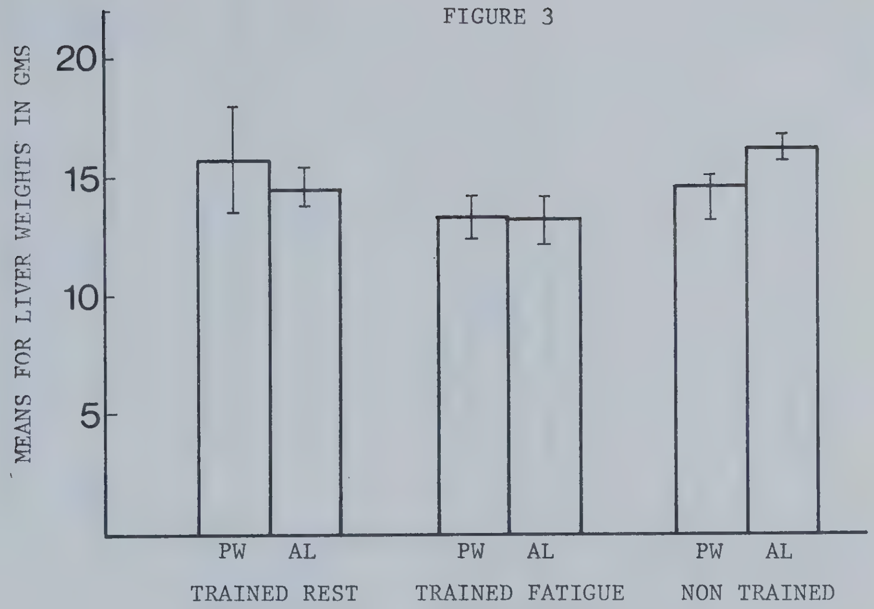


FIGURE 4

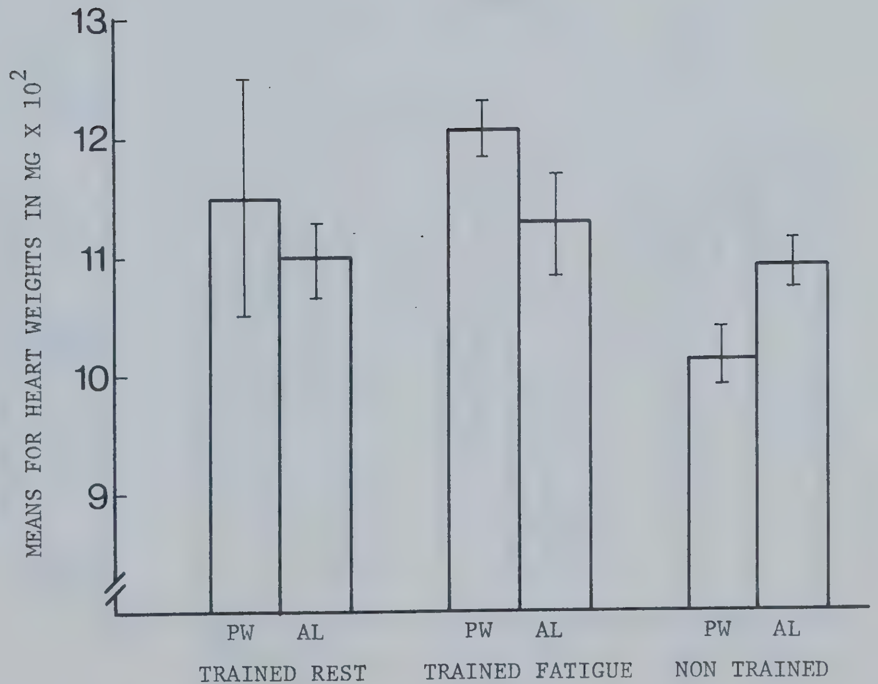


FIGURE 5

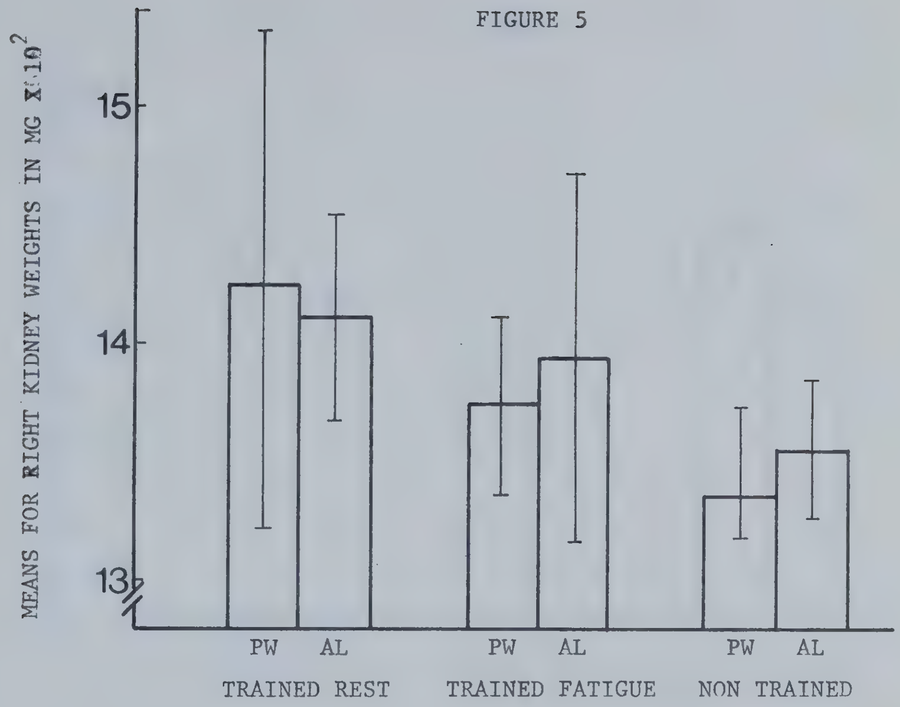


FIGURE 6

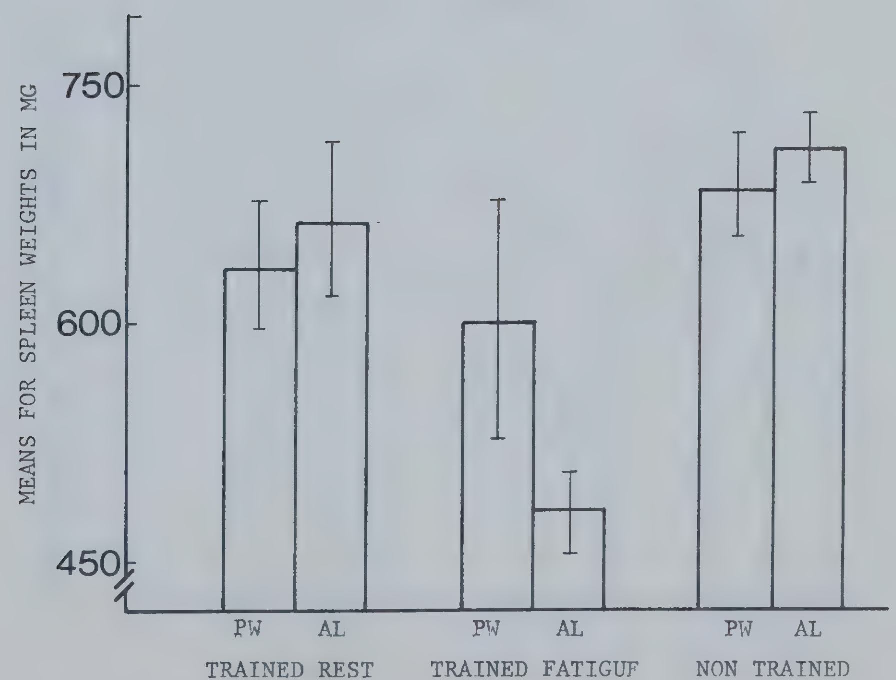


FIGURE 7

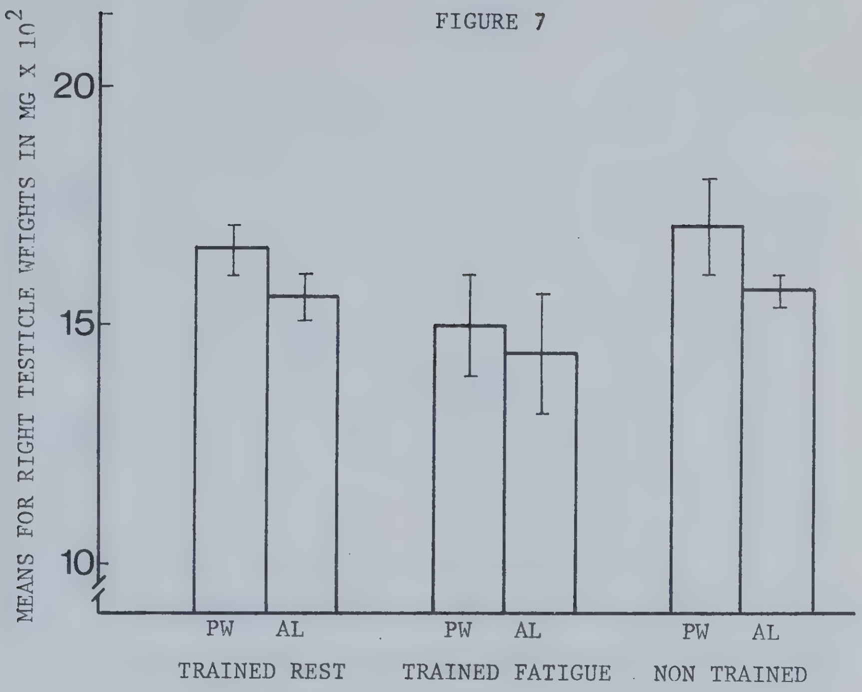


FIGURE 8

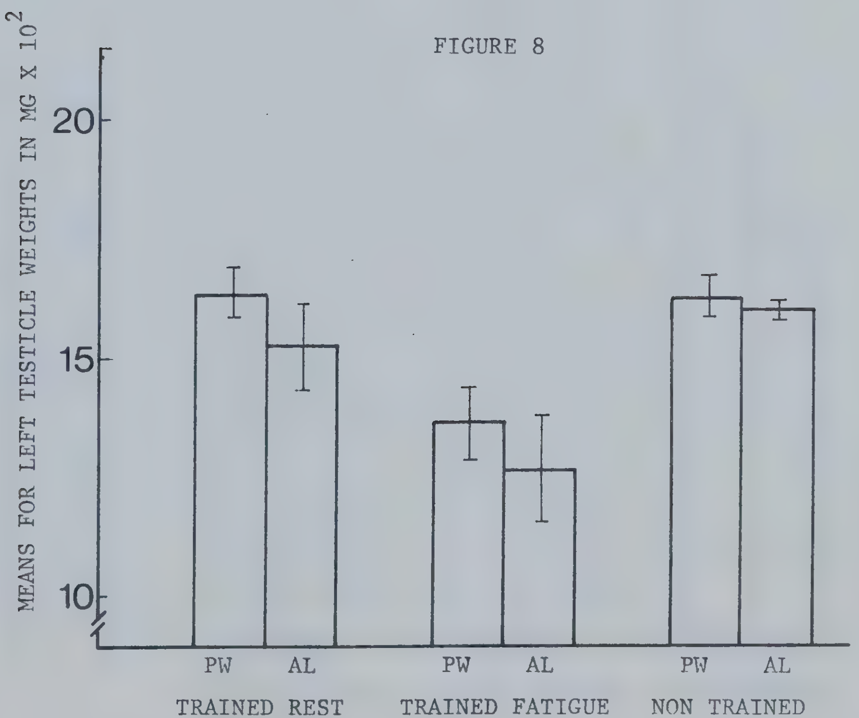


FIGURE 9

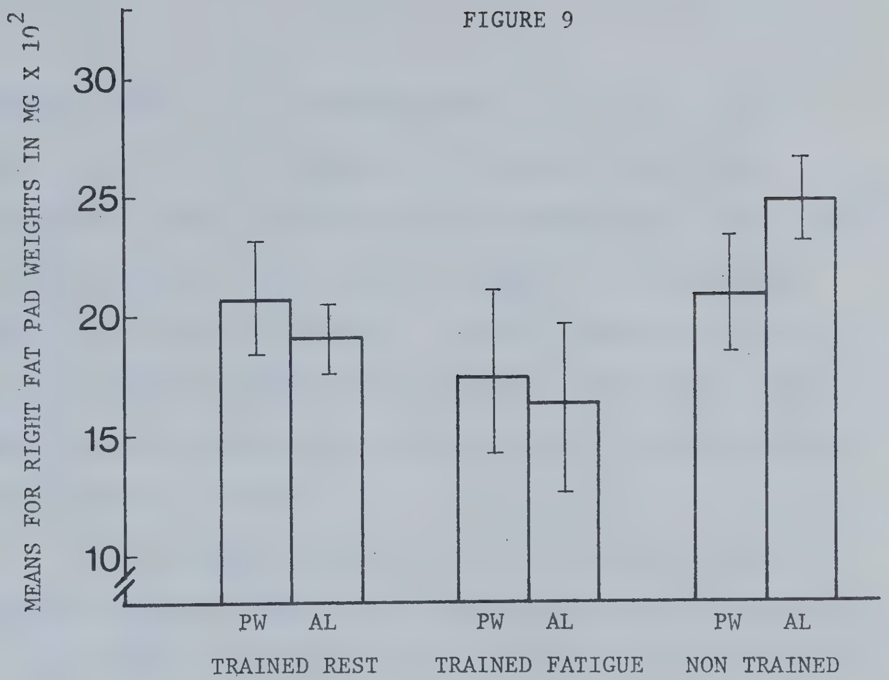
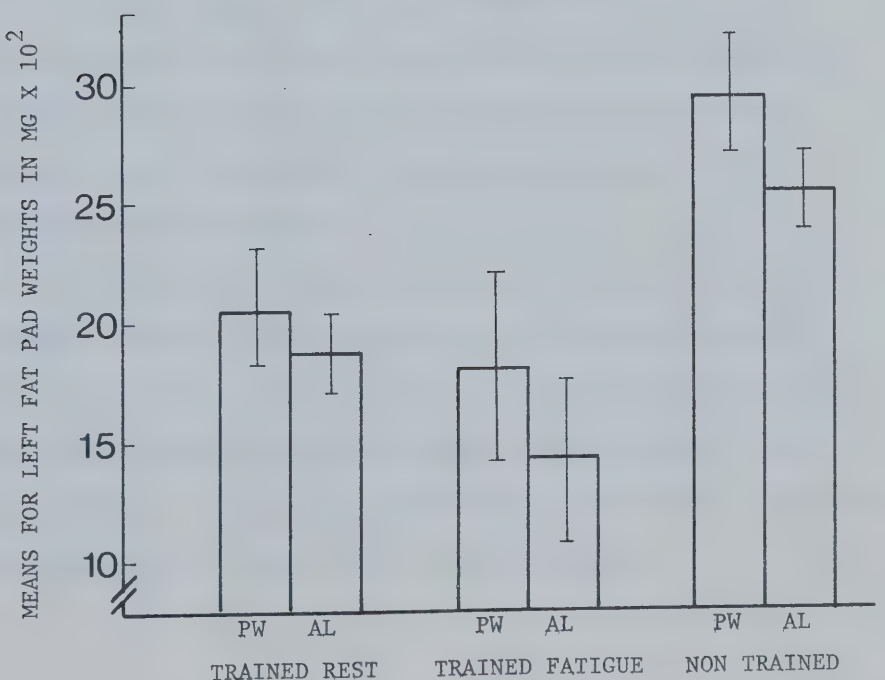


FIGURE 10



(Figures 11 and 12). All trained animals had similar adrenal weights. Only the right adrenals of fatigued animals were significantly larger than those of the control group. Diet showed significant effects only on adrenal weight, with pair-weighed animals having lighter adrenals. A slight interaction of diet and exercise was demonstrated for heart weight. Gastrocnemius and biceps brachii muscle weights were uneffected by either exercise or diet (Figures 13 to 15).

Liver glycogen stores of resting trained and control groups were similar (Figure 16). Animals exercised to exhaustion had significantly reduced glycogen stores when compared to animals sacrificed at rest. Diet did not effect liver glycogen storage.

Skeletal muscle glycogen levels of resting trained and control groups were similar (Figures 17 and 18). No significant differences among exercise groups for gastrocnemius and bicep glycogen content were found. Pair-weighting did not effect muscle carbohydrate storage.

No differences existed between resting trained or control groups for plasma or muscle lactate levels (Figures 19 and 20). Exhaustive exercise caused a significant increase in blood lactate levels above resting and control values. Muscle lactate levels at fatigue showed a significant decrease from control values. Dietary restriction did not effect blood or muscle lactates.

FIGURE 11

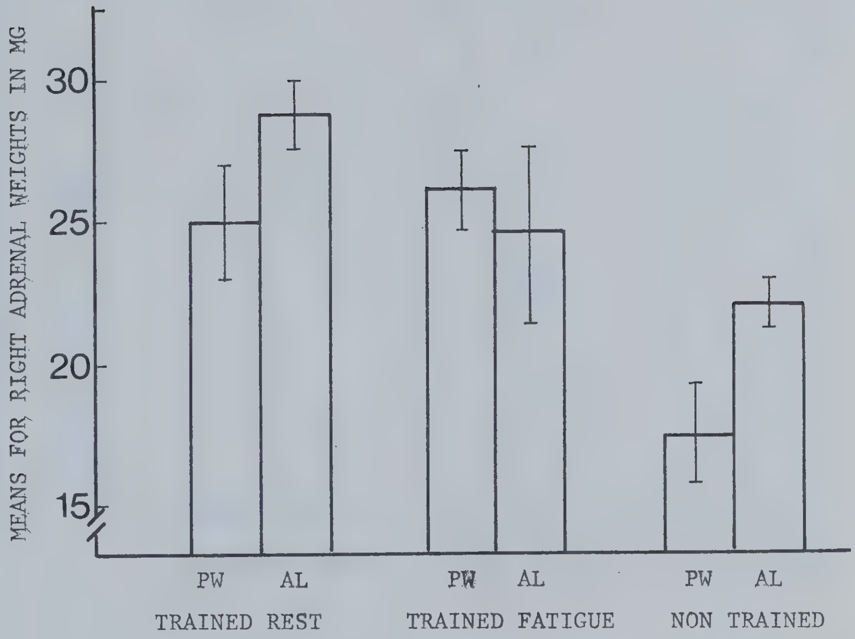


FIGURE 12

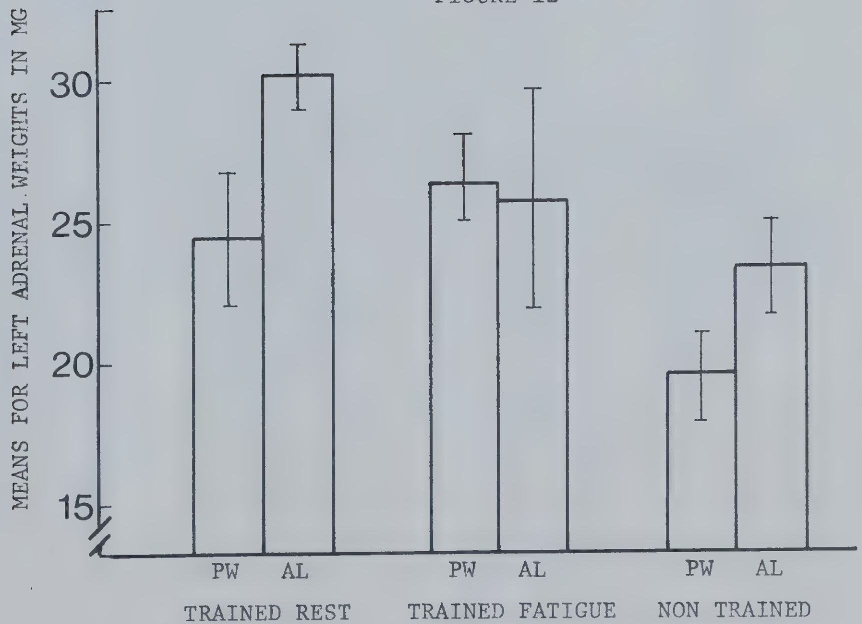


FIGURE 13

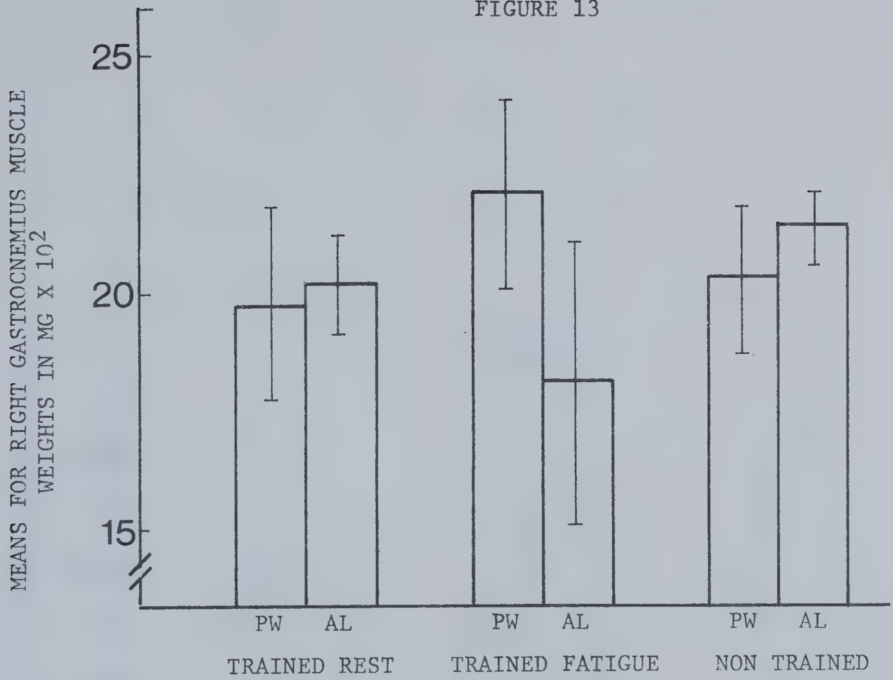


FIGURE 14

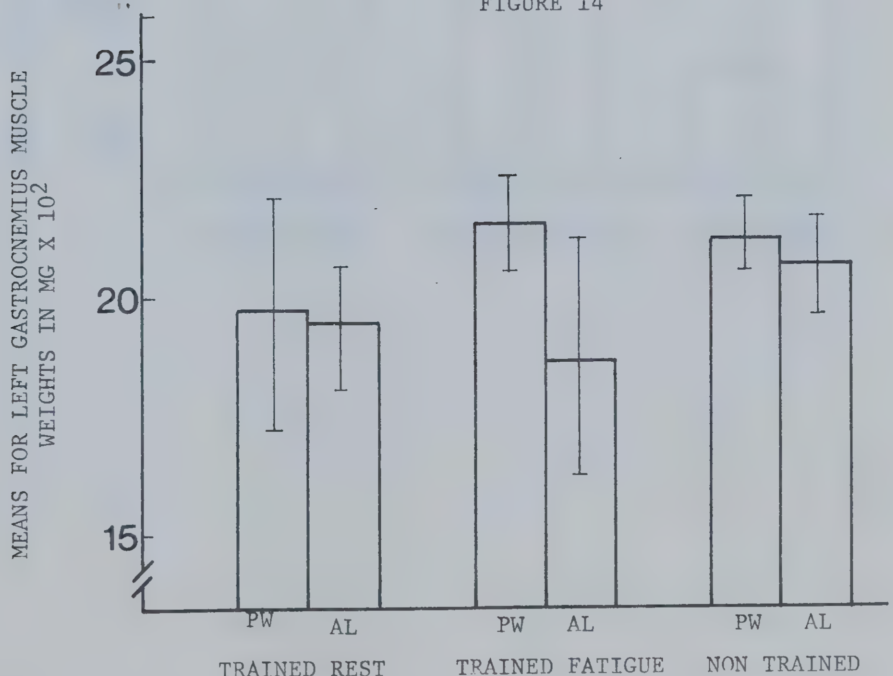


FIGURE 15

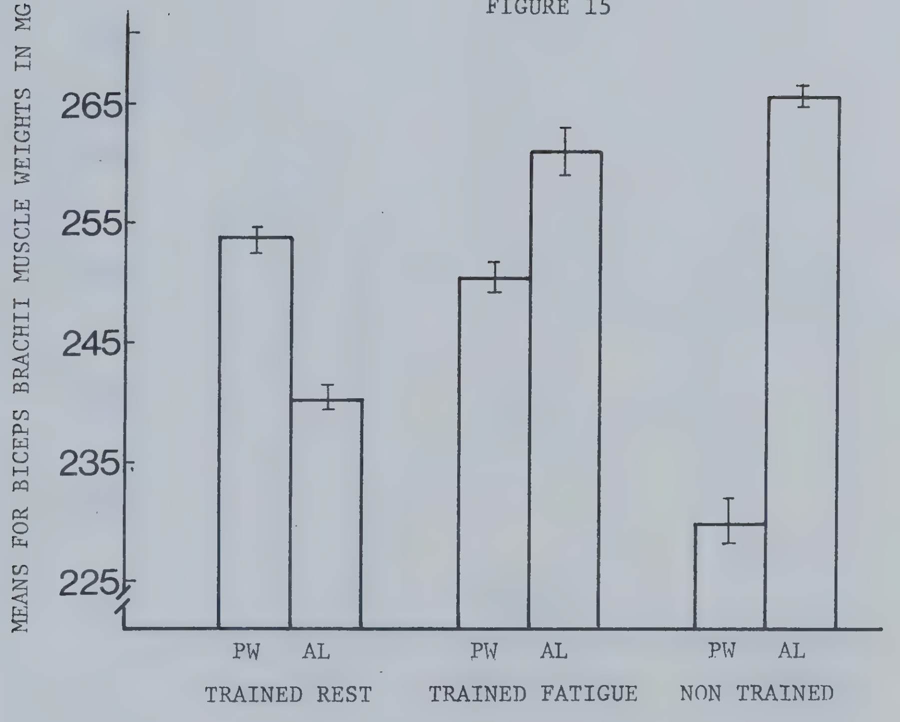

MEANS FOR BICEPS BRACHII MUSCLE WEIGHTS IN MG
265
255
245
235
225
PW
AL
PW
AL
PW
AL
TRAINED REST
TRAINED FATIGUE
NON TRAINED

FIGURE 16

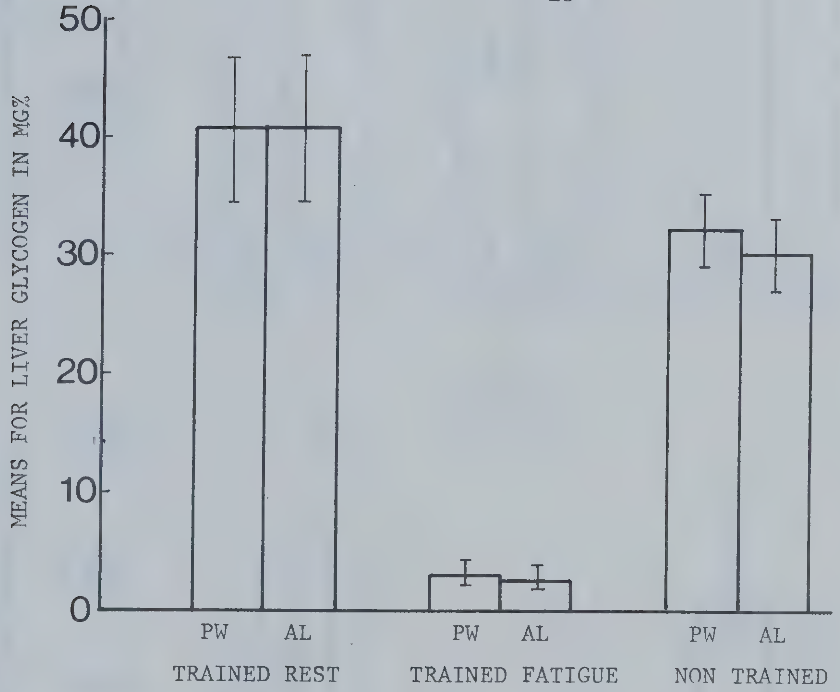

MEANS FOR LIVER GLYCOGEN IN MG%
50
40
30
20
10
0
PW
AL
TRAINED REST
PW
AL
TRAINED FATIGUE
PW
AL
NON TRAINED

FIGURE 17

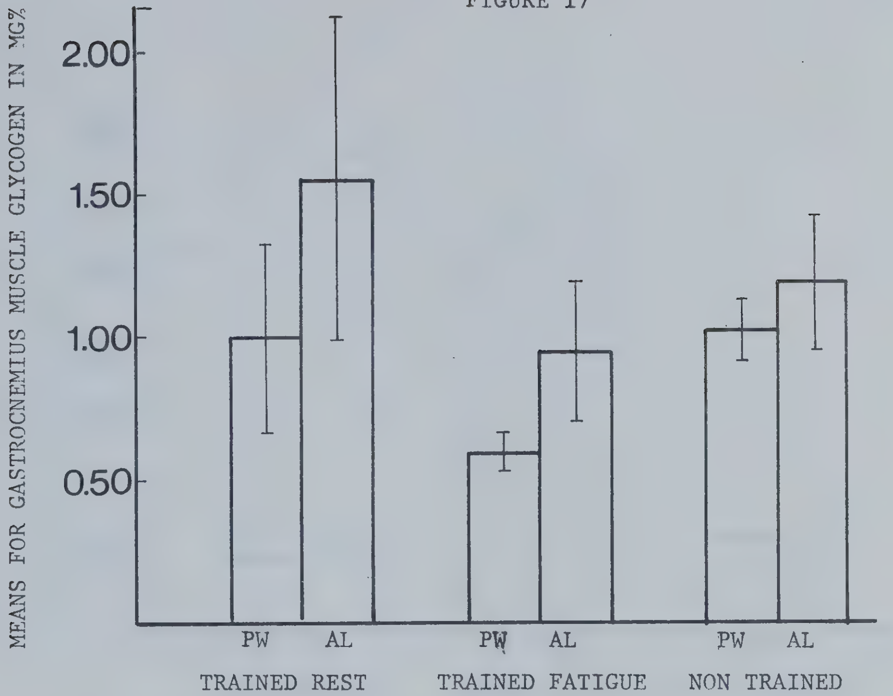


FIGURE 18

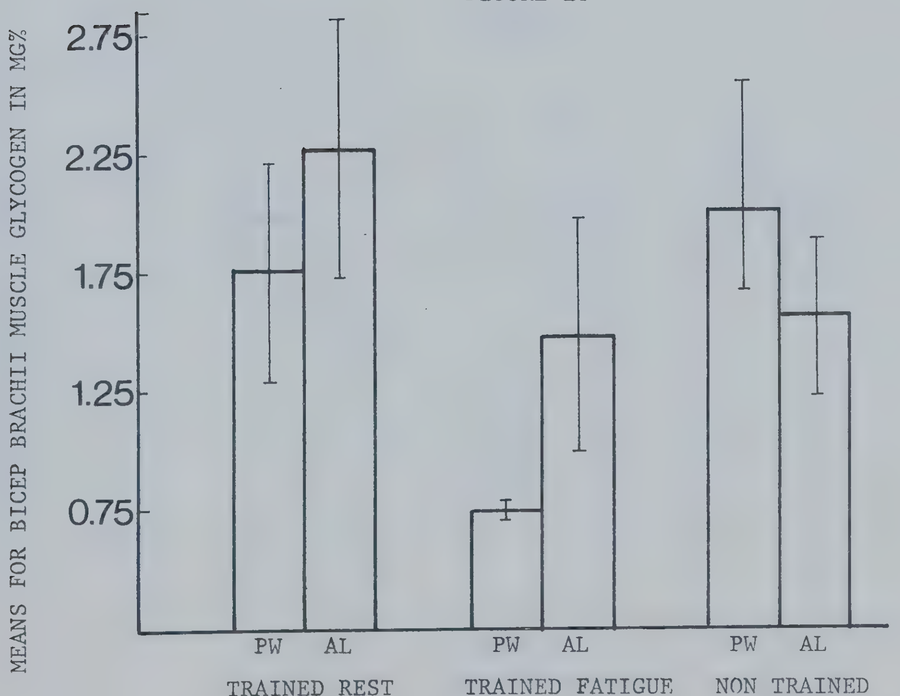


FIGURE 19

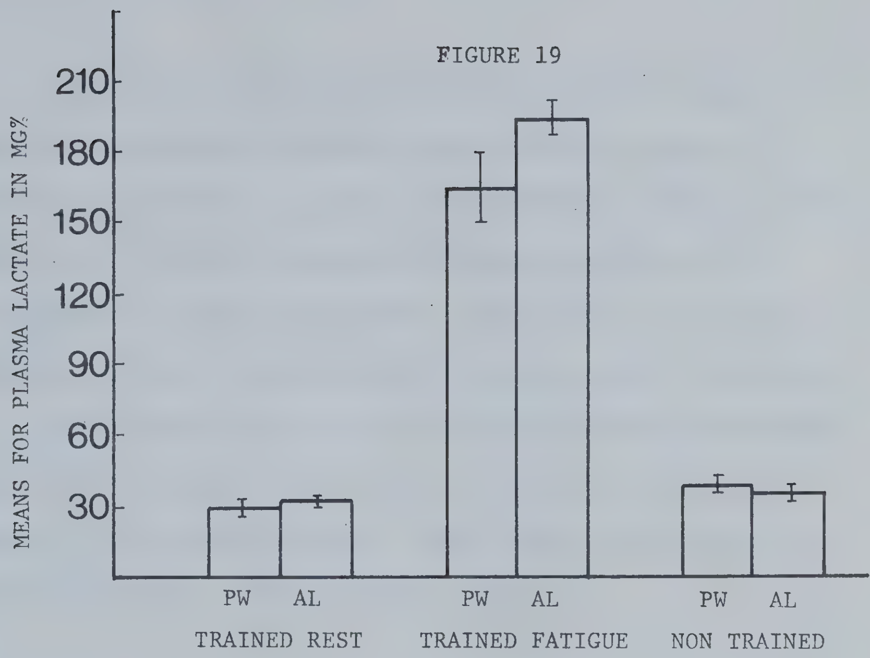
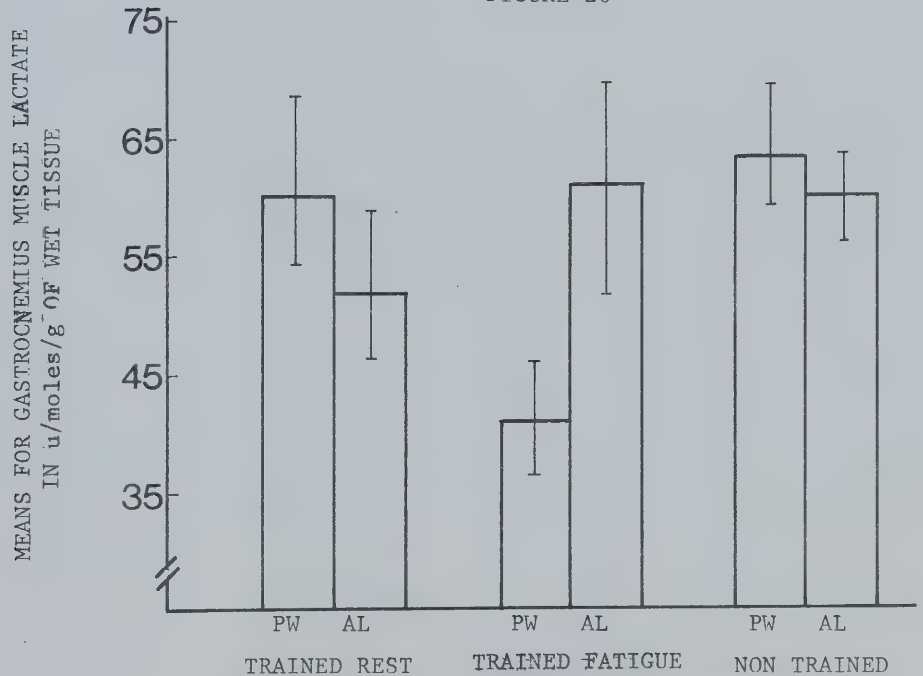


FIGURE 20



Plasma and adipose tissue FFA concentrations showed no significant differences between resting trained animals and sedentary controls (Figures 21 to 23). Fatiguing exercise produced a significant increase in plasma FFA concentration and the concentration of adipose tissue FFA. Diet did not produce any significant effect on either plasma or adipose tissue FFA levels.

Blood glucose levels were similar for resting trained and control animals (Figure 24). Exercise caused a significant decrease in blood glucose levels. Controlled diet did not have any apparent effect on blood glucose levels.

FIGURE 21

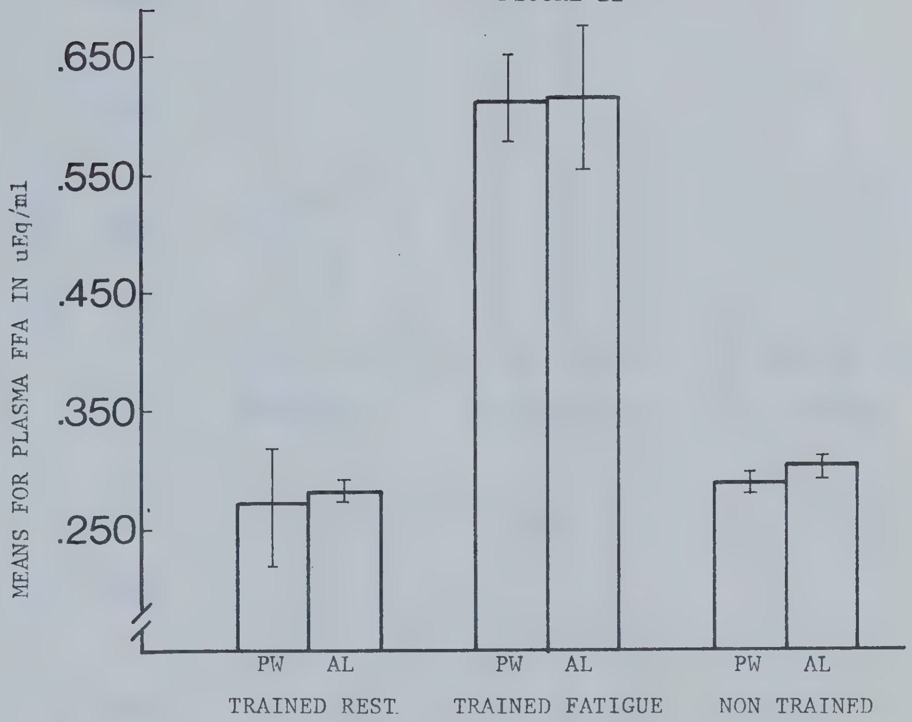

MEANS FOR PLASMA FFA IN uEq/ml
.650
.550
.450
.350
.250
PW
AL
PW
AL
PW
AL
TRAINED REST
TRAINED FATIGUE
NON TRAINED

FIGURE 22

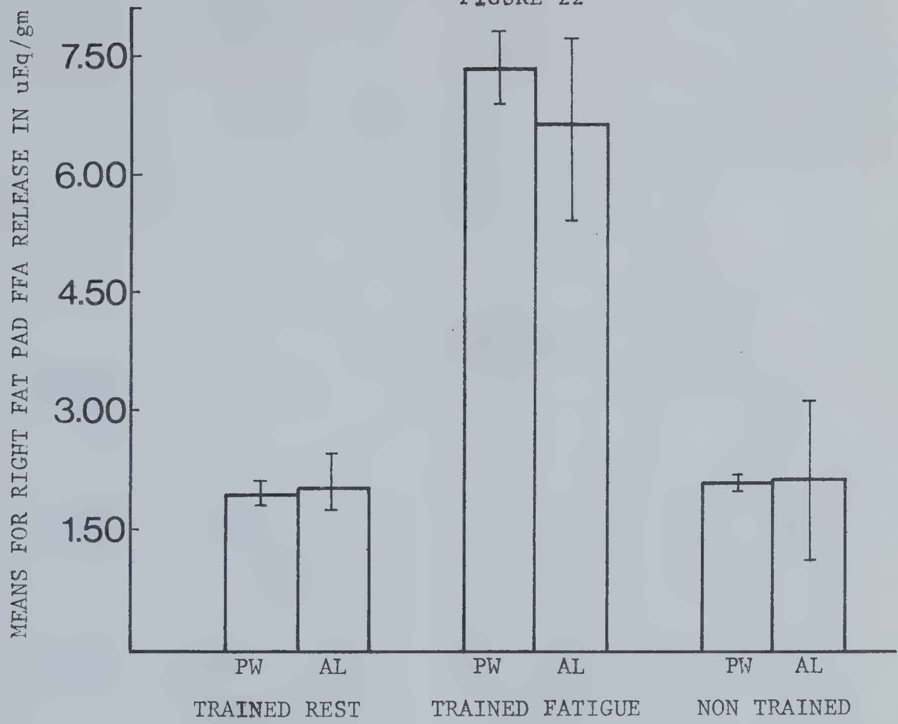


FIGURE 23

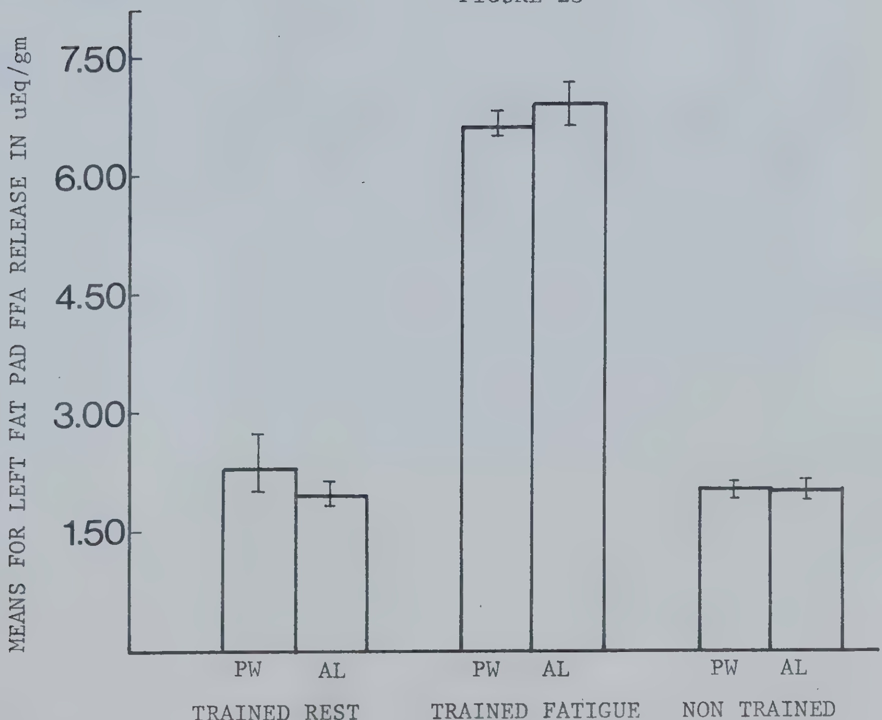
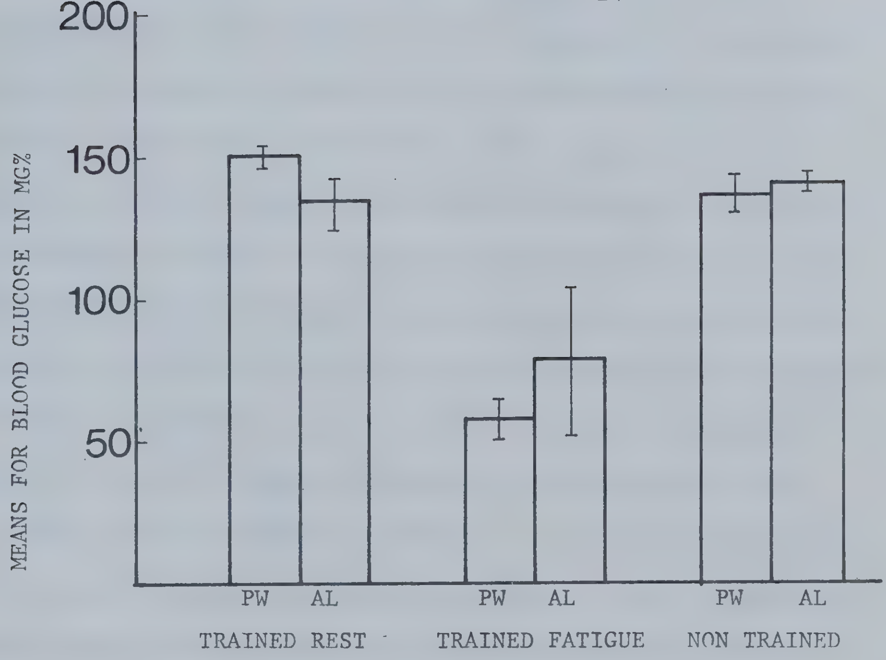


FIGURE 24



200
150
100
50
MEANS FOR BLOOD GLUCOSE IN MG%
PW
AL
PW
AL
PW
AL
TRAINED REST
TRAINED FATIGUE
NON TRAINED

CHAPTER V

DISCUSSION

DIET

Rats engaged in a regular training program show a significantly lesser weight gain when compared to "free-eating" sedentary controls (Steinhaus et al, 1932; Kimeldorf and Baum, 1954; Dalhgren, 1965; Bloor et al, 1968; Crews et al, 1969; Buuck and Tharp, 1971). This inhibited weight gain is generally attributed to the increased caloric expenditure as well as the depressant effect of exercise on appetite (Crews et al, 1969; Oscai et al, 1971a; Oscai et al, 1972). For purposes of comparing trained and untrained animals without the intervening variable of these total body weight differences recent researchers have turned to a pair-weighing technique. The results of the present study show that pair-weighing, in addition to being effective in maintaining equal body weights between trained and untrained animals, also resulted in similar resting organ and muscle weights, and plasma and tissue metabolites. Adrenal weights of pair-weighed animals were lighter than those of the ad libitum fed animals. This effect of diet on adrenal weights was considered to be attributable to problems in isolating and weighing the excised tissues of the ad lib sedentary rats that were apparently fatter. Therefore, using the described training program, the advantages of employing pair-weighed animals versus sedentary "free-eating"

controls were not borne out. On the other hand, the limitations of pair-weighing in terms of severe alterations of body weight and composition were not elucidated. Other investigators using more intensive training regimes showed larger differences in body weight between trained and untrained "free-eaters" and nontrained pair-weighed rats than the present study, and reported marked changes in fat and protein composition in the sedentary pair-weighed animals (Crews et al, 1969; Oscai et al, 1972). In the present study, had the exercise intensity been greater, the increased dietary restriction of the pair-weighed animals may have resulted in more significant body and organ weight differences from those fed ad libitum, thus contraindicating their absolute comparison with trained animals.

TRAINING

The lighter fat pad weights of the trained rats found in the present study are concurrent with previously reported adipose changes in exercised animals. Crews et al (1969) noted that exercised animals were leaner than sedentary controls and attributed this to the regular lipid mobilizing effect of exercise in addition to changes in caloric expenditure and intake. The adrenal hypertrophy with training is in agreement with the findings of other investigators (Kimeldorf and Baum, 1954; Gollnick, Struck and Bogyo, 1967; Buuck and Tharp, 1971): the 30% increase in adrenal weight

with training is comparable to the 35% increase found by Jones et al (1964) using a strenuous swimming program.

The fact that no other organ weight changes were noted with training in spite of previously observed changes in liver (Hatai, 1915; Jones et al, 1964), kidney (Hatai, 1915; Kimeldorf and Baum, 1954; Gollnick and Hearn, 1961; Bloor et al, 1968), and testicular weights (Hatai, 1915; Steinhaus et al, 1932; Tipton et al, 1968) suggests the possible effect of different training regimes on these parameters. The spleen weights of trained and untrained animals were similar and concur with the observations of Tipton et al (1966) and Bloor et al (1968) since spleen weight appears to be dependent on the severity of immediate exercise on the animal rather than the training.

The lack of significant differences in heart weights between trained and untrained rats was unexpected in light of the marked cardiac hypertrophy observed with training by many investigators (Hatai, 1915; Donaldson and Meeser, 1932; Kimeldorf and Baum, 1954; Van Liere et al, 1965; Van Liere and Northup, 1967). Exercised animals in the present study had heart weights that were 13.3% greater than their pair-weighed controls. The combination of the pair-weighed and ad libitum nontrained animals may have masked any trends in this direction.

The results of this study indicate that training has little effect upon the storage of liver or skeletal muscle glycogen.

Gollnick et al (1970) and Fielding (1973) found no significant training effects for skeletal muscle glycogen, however, their values are 50% higher than those reported in the present study. This discrepancy may be related to the high variance and low N of the present study. Similarly, blood glucose levels are not effected by training. A hyperglycemia was noted when comparing the present results with those previously reported (Fielding, 1973; Taylor and Rao, 1973). This hyperglycemic response can be primarily attributed to the hypoxia that may have resulted from the ether anaesthesia. Hypoxia is known to cause an increase in blood glucose levels by stimulating glycogenolysis resulting in an increased hepatic glucose output (Rowell et al, 1968).

The findings of the present study for plasma and muscle lactate levels, that trained and untrained rats have similar resting values, concur with those of Fielding (1973) and Taylor and Rao (1973), however, our values for plasma lactates are slightly higher as are the values for muscle lactates than those cited in the literature. Again, the hypoxia produced by over anaesthetizing animals with ether could be responsible for the higher lactate levels observed. The variance within groups for gastrocnemius muscle lactate concentration may have also contributed to the discrepancies observed, since it was noted to be high.

In previous studies and in the present work no training effects have been noted for resting plasma FFA levels (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1972; Fielding, 1973) or

adipose tissue FFA concentrations (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1972; Fielding, 1973).

EXHAUSTIVE EXERCISE

The lighter testicular weights found are in agreement with the results of Tipton et al (1968) and Steinhaus et al (1932); however, the rationale for such an occurrance is still unknown.

The specific lipid-mobilizing effect of prolonged work suggests a primary reason for the finding of lighter fat pads in exhausted animals. A definite color difference (off-white to pink) between the fat pads of rats sacrificed at rest versus those sacrificed after an exhaustive exercise bout was noted. This was interpreted as an increased perfusion of the area as part of the increased mobilization of FFA in the adipose tissue. Both plasma and adipose tissue FFA levels increase as a result of prolonged exercise (Gollnick, 1967; Gollnick et al, 1970; Taylor, 1972; Fielding, 1973) and the increased mobilization of lipid stores would result in a decreased adiposity.

The decreased liver glycogen content with exhaustive exercise noted in this study concurs with the work of other researchers (Ahlborg et al, 1967; Gollnick and King, 1969; Gollnick et al, 1970; Costill et al, 1971; Karlsson and Saltin, 1971). During prolonged exercise the decreased liver glycogen reflects an increased glycogenolysis in the liver in an attempt to maintain the slowly decreasing blood sugar levels, such as those observed in this and

other studies (Ahlborg et al, 1967; Gollnick et al, 1970; Taylor et al, 1971; Taylor and Rao, 1973).

Numerous investigators have attempted to relate initial muscle glycogen content and performance time during prolonged submaximal work to exhaustion. The data from the present study does not lend support to either the enhanced glycogen storage in the trained muscles or its depletion with prolonged work. However, because of the large variation in the data, it is felt that further research is warrented using a larger number of experimental animals and possibly another assay technique. As well, there may be a species difference in the muscles utilized for running and the ability of these muscles to store carbohydrates.

The increased plasma lactate concentrations found in the fatigued rats are not consistent with the values reported by others at the end of prolonged exhaustive work for humans (Costill, 1970; Astrand et al, 1963) or rats (Taylor and Rao, 1973; Fielding, 1973). It is proposed that the possible hypoxia induced by the ether anaesthesia was responsible for the increased plasma lactate levels observed in this study. In addition, even though the expected changes occurred in plasma and tissue FFA and plasma glucose concentrations at fatigue, (depleted glycogen) in liver, the possibility that hypoxia resulting from the anaesthesia was responsible for these changes must also be entertained. In all liklihood, the final minutes of the severe exercise may have been hypoxic thus resulting in anaerobic catabolism. The failure in the

present study for muscle lactate levels to reflect the same changes as the plasma lactate concentrations at fatigue were unexpected since plasma lactate levels have been noted to be dependent on tissue lactate concentrations (Dawson et al, 1971; Knuttgen and Saltin, 1972; Taylor and Rao, 1973; Fielding, 1973).

Although present results for muscle lactates follow the expected trends to approximate resting values following extended exercise to fatigue, the hypoxia produced by the anaesthetic anaerobic metabolism at the end of work or a combination of the two could have resulted in the higher plasma lactate levels by an inhibition of the oxidation of lactate in the liver via the Cori cycle.

CHAPTER VI

SUMMARY AND CONCLUSIONS

SUMMARY

Sixty-two male Wistar rats were divided into pair-weighed and ad libitum dietary groups for purposes of evaluating the effect of pair-weighing on the storage and utilization of energy sources. Half of each group were engaged in a moderate treadmill training program consisting of running at 1.0 m.p.h. for one hour per day, five successive days per week, for ten to fourteen weeks.

The results indicated that pair-weighing did not significantly effect organ or muscle weights, with the exception of adrenal weights and that plasma and tissue metabolites were not effected. Training caused a marked increase in adrenal weights and a decrease in epididymal fat pad weights. All other organ and muscle weights, and plasma and tissue metabolites remained unchanged. Exhaustive exercise produced a reduction in liver glycogen content, and blood glucose levels, and increased plasma and adipose tissue FFA and plasma lactate levels.

CONCLUSION

Within the limitations of this study, pair-weighing did not significantly effect the storage or utilization of energy sources.

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APPENDIX A

STANDARD CURVES

FIGURE 25

STANDARD CURVE FOR GLYCOGEN

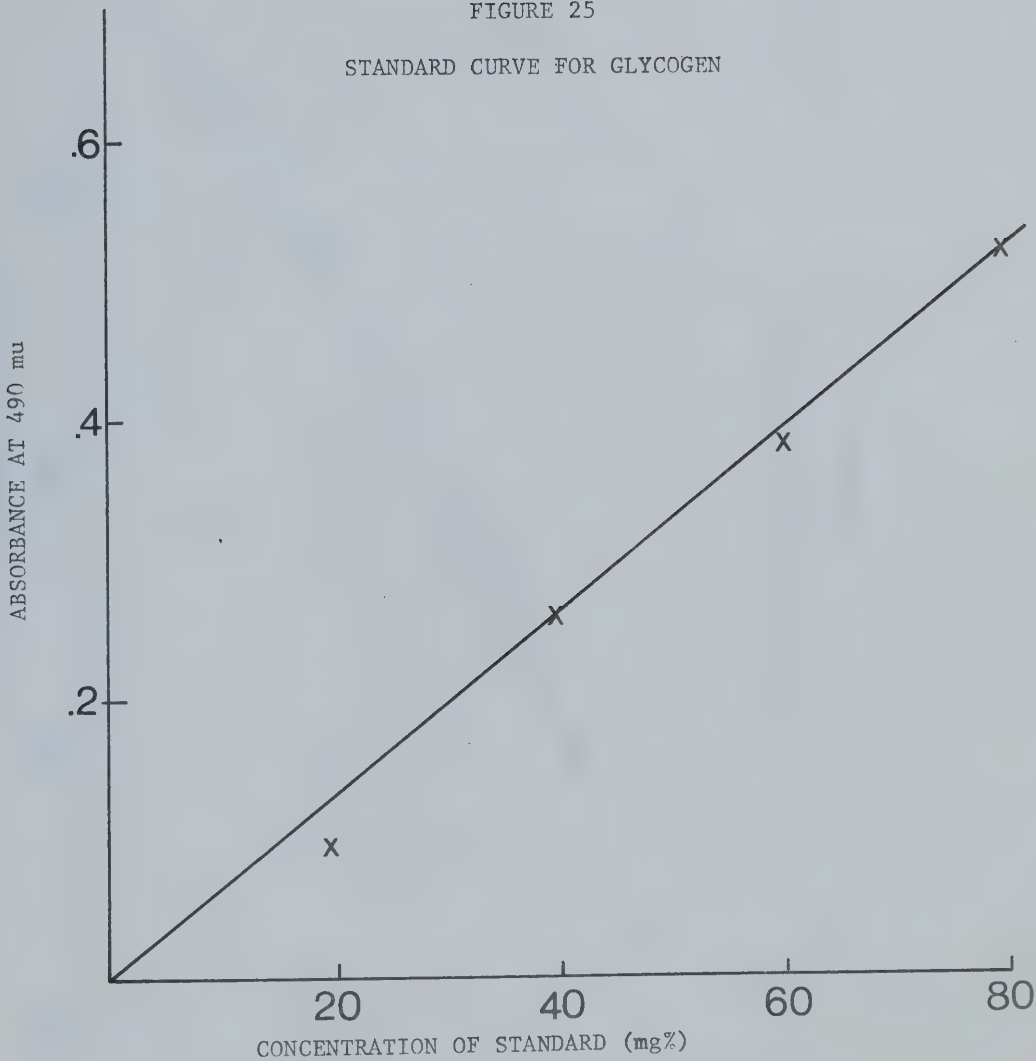


FIGURE 26

STANDARD CURVE FOR LACTIC ACID

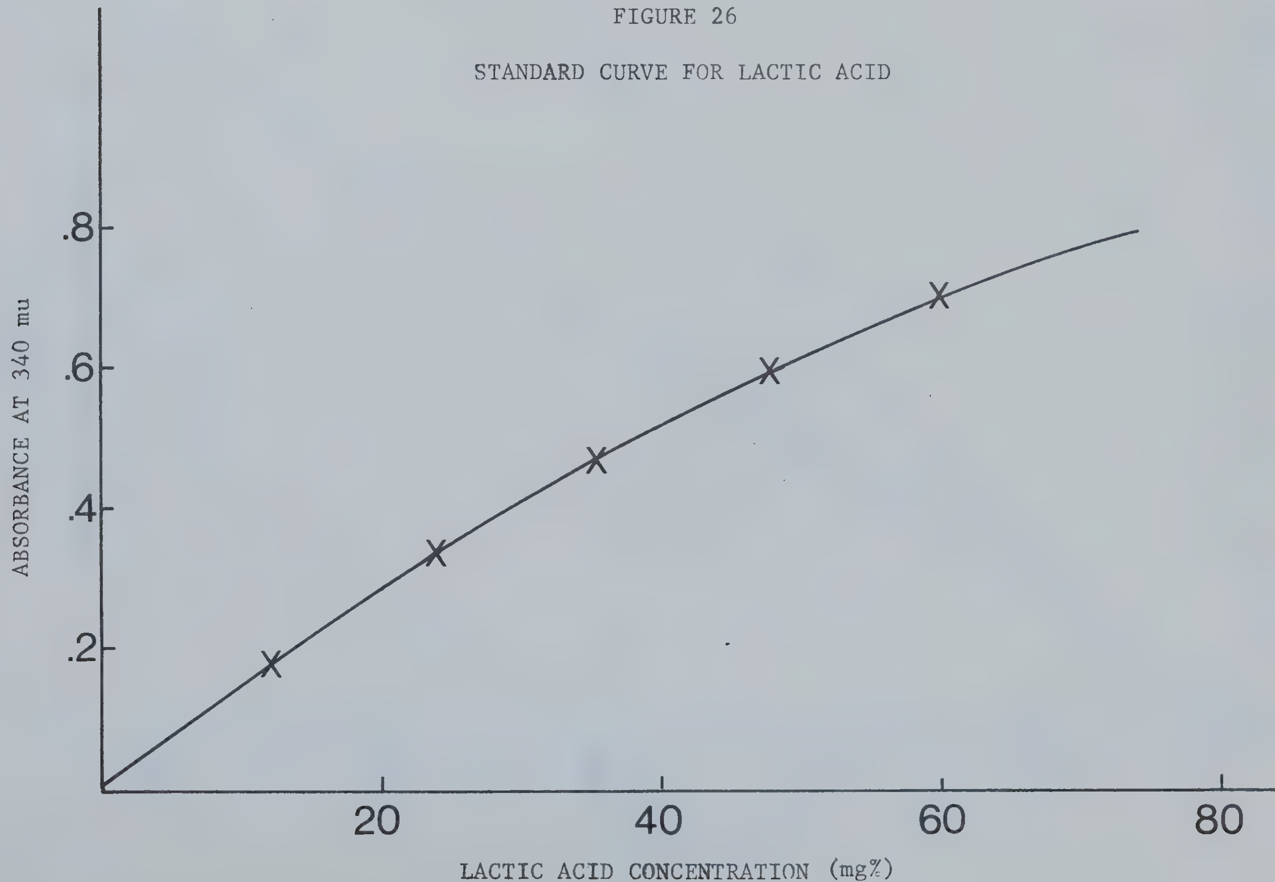


FIGURE 27

STANDARD CURVE FOR FREE FATTY ACIDS

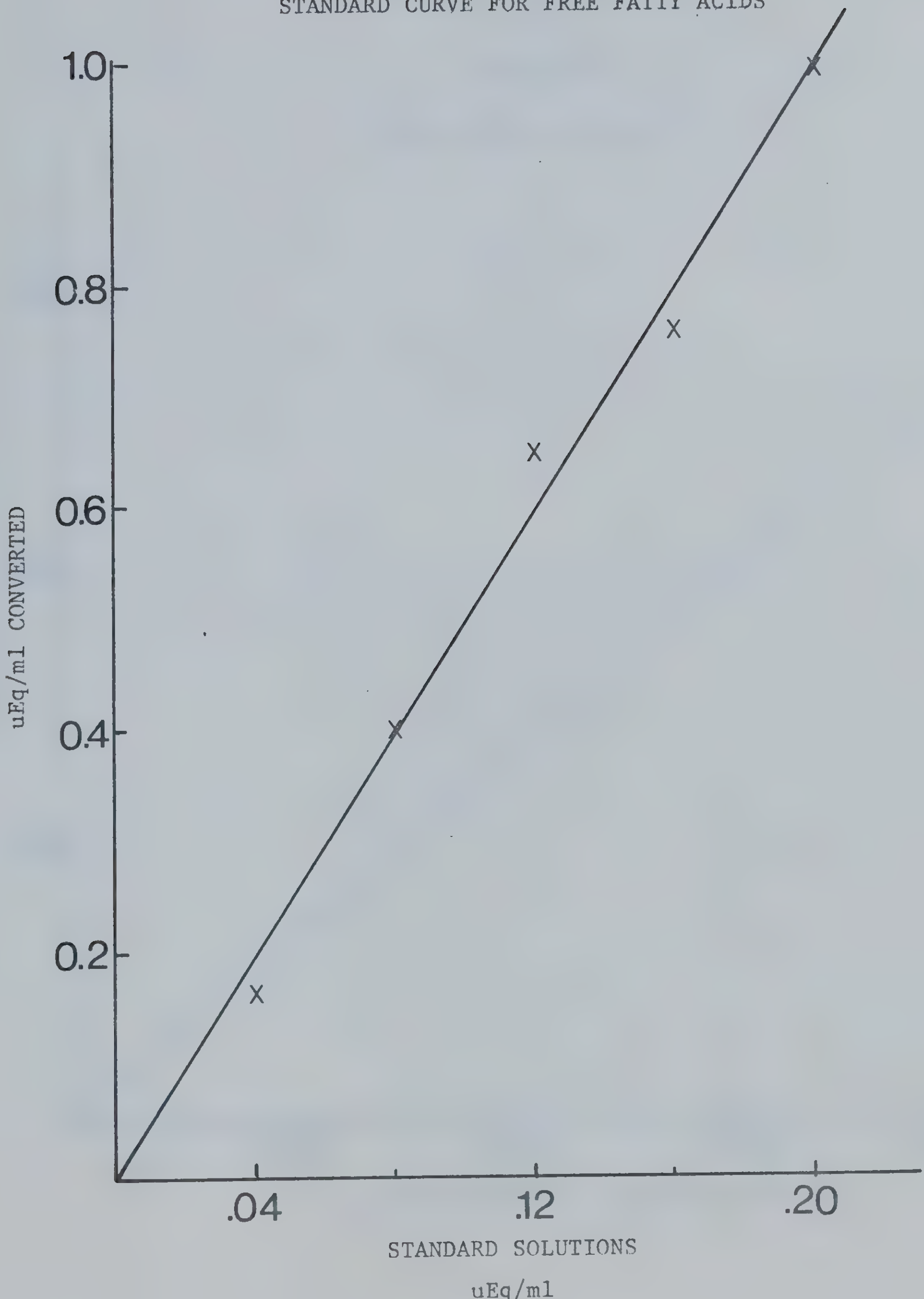
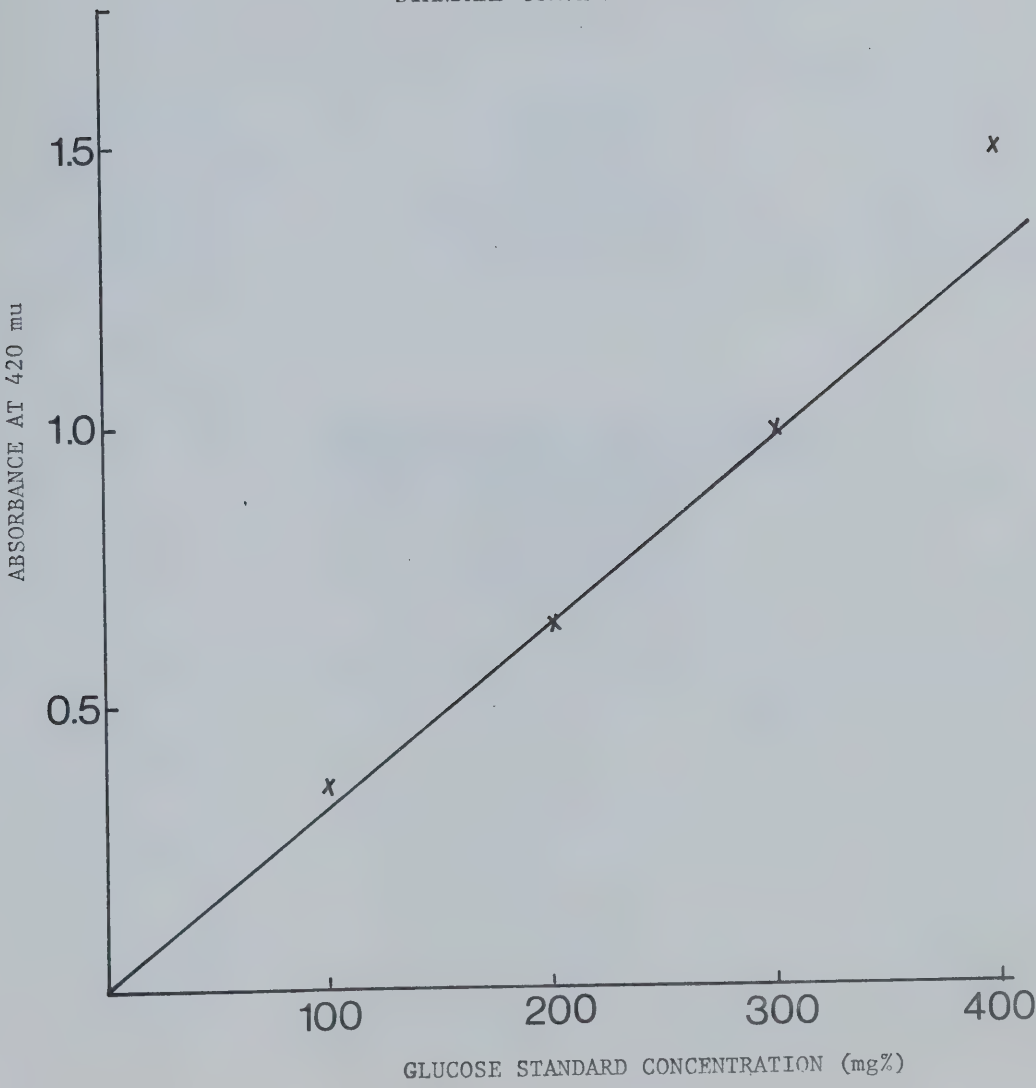


FIGURE 28

STANDARD CURVE FOR GLUCOSE



APPENDIX B

TABLE OF MEANS $\pm$ S.E.M.

LEGEND FOR ALL TABLES IN APPENDIX B

PW - PAIR-WEIGHED

AL - AD LIBITUM

TABLE 1

MEANS FOR TOTAL BODY WEIGHT
IN GMS.

Group	Diet	
	PW	AL
Trained Rest	450.0 ± 25.5	416.6 ± 15.1
Trained Fatigue	439.0 ± 11.4	415.4 ± 37.5
Non-Trained	445.9 ± 11.4	471.5 ± 11.0

TABLE 2

MEANS FOR LIVER WEIGHTS
IN GMS.

Group	Diet	
	PW	AL
Trained Rest	15.8 ± 2.2	14.7 ± 0.8
Trained Fatigue	13.4 ± 0.8	13.3 ± 1.0
Non-Trained	14.8 ± 0.5	16.4 ± 0.5

TABLE 3

MEANS FOR HEART WEIGHTS
IN MG.

Group	Diet PW	AL
Trained Rest	1151.2 ± 103.4	1101.2 ± 34.3
Trained Fatigue	1209.7 ± 26.9	1128.0 ± 46.4
Non-Trained	1016.4 ± 24.4	1095.6 ± 22.4

TABLE 4

MEANS FOR RIGHT KIDNEY WEIGHTS
IN MG.

Group	Diet PW	AL
Trained Rest	1426.5 ± 105.4	1411.4 ± 43.1
Trained Fatigue	1374.4 ± 37.2	1393.7 ± 77.8
Non-Trained	1347.3 ± 27.4	1356.5 ± 28.8

TABLE 5

MEANS FOR SPLEEN WEIGHTS
IN MG.

Group	Diet: PW	Diet: AL
Trained Rest	638.5 ± 40.0	667.0 ± 48.8
Trained Fatigue	603.2 ± 76.7	484.1 ± 24.1
Non-Trained	688.6 ± 38.1	713.4 ± 21.6

TABLE 6

MEANS FOR RIGHT ADRENAL WEIGHTS
IN MG.

Group	Diet	
	PW	AL
Trained Rest	25.0 $\pm$ 2.1	28.8 $\pm$ 1.2
Trained Fatigue	26.1 $\pm$ 1.3	24.6 $\pm$ 3.2
Non-Trained	17.5 $\pm$ 1.8	22.6 $\pm$ 1.3

TABLE 7

MEANS FOR LEFT ADRENAL WEIGHTS
IN MG.

Group	Diet	
	PW	AL
Trained Rest	24.4 $\pm$ 2.3	30.4 $\pm$ 1.1
Trained Fatigue	26.6 $\pm$ 1.6	25.8 $\pm$ 4.0
Non-Trained	19.6 $\pm$ 1.8	23.8 $\pm$ 1.2

TABLE 8

MEANS FOR RIGHT TESTICLE WEIGHTS
IN MG.

Group	Diet	
	PW	AL
Trained Rest	1662.8 ± 58.4	1559.7 ± 52.8
Trained Fatigue	1501.7 ± 104.5	1448.1 ± 131.2
Non-Trained	1732.0 ± 105.0	1587.2 ± 42.1

TABLE 9

MEANS FOR LEFT TESTICLE WEIGHTS
IN MG.

Group	Diet	
	PW	AL
Trained Rest	1644.3 ± 52.6	1533.5 ± 92.6
Trained Fatigue	1370.3 ± 77.8	1269.3 ± 111.6
Non-Trained	1636.8 ± 41.1	1606.5 ± 23.8

TABLE 10

MEANS FOR RIGHT FAT PAD WEIGHTS
IN MG.

Group	Diet	
	PW	AL
Trained Rest	2077.2 ± 239.9	1907.7 ± 148.5
Trained Fatigue	1760.0 ± 345.0	1632.3 ± 357.1
Non-Trained	2098.0 ± 242.9	2499.7 ± 181.2

TABLE 11

MEANS FOR LEFT FAT PAD WEIGHTS
IN MG.

Group	Diet	
	PW	AL
Trained Rest	2068.1 ± 251.8	1880.3 ± 158.1
Trained Fatigue	1812.1 ± 399.7	1429.5 ± 357.3
Non-Trained	2963.7 ± 241.3	2560.0 ± 160.8

TABLE 12

MEANS FOR RIGHT GASTROCNEMIUS
MUSCLE WEIGHTS IN MG.

Group	Diet PW	AL
Trained Rest	1987.9 ± 200.6	2033.8 ± 106.0
Trained Fatigue	2220.8 ± 200.6	1829.2 ± 291.3
Non-Trained	2043.2 ± 154.8	2148.6 ± 83.4

TABLE 13

MEANS FOR LEFT GASTROCNEMIUS
MUSCLE WEIGHTS IN MG.

Group	Diet PW	AL
Trained Rest	1980.9 ± 242.4	1950.1 ± 135.1
Trained Fatigue	2158.9 ± 100.0	1884.9 ± 258.0
Non-Trained	2147.3 ± 86.5	2076.3 ± 108.9

TABLE 14

MEANS FOR BICEPS BRACHII MUSCLE WEIGHTS IN MG.

Group	Diet	
	PW	AL
Trained Rest	253.8 ± 1.0	240.4 ± 1.0
Trained Fatigue	250.7 ± 1.3	261.0 ± 2.1
Non-Trained	229.9 ± 1.8	265.9 ± 1.0

TABLE 15

MEANS FOR LIVER GLYCOGEN IN MG. %

Group	Diet	
	PW	AL
Trained Rest	41.5 ± 6.0	41.2 ± 5.6
Trained Fatigue	3.4 ± 0.9	3.1 ± 1.0
Non-Trained	32.6 ± 2.9	30.5 ± 3.1

TABLE 16

MEANS FOR GASTROCNEMIUS MUSCLE GLYCOGEN IN MG %

Group	Diet	
	PW	AL
Trained Rest	1.01 ± 0.36	1.57 ± 0.58
Trained Fatigue	0.59 ± 0.06	0.95 ± 0.25
Non-Trained	1.06 ± 0.12	1.22 ± 0.24

TABLE 17

MEANS FOR BICEPS BRACHII MUSCLE GLYCOGEN IN MG %

Group	Diet	
	PW	AL
Trained Rest	1.76 ± 0.46	2.28 ± 0.55
Trained Fatigue	0.75 ± 0.05	1.48 ± 0.50
Non-Trained	2.13 ± 0.43	1.58 ± 0.31

TABLE 18

MEANS FOR PLASMA LACTATE
IN MG. %

Group	Diet: PW	Diet: AL
Trained Rest	30.0 ± 3.5	32.6 ± 2.0
Trained Fatigue	165.8 ± 15.0	193.3 ± 6.7
Non-Trained	38.8 ± 3.5	35.5 ± 3.6

TABLE 19

MEANS FOR GASTROCNEMIUS MUSCLE
LACTATE IN u MOLES/G

Trained Rest	61.8 ± 7.1	52.8 ± 6.3
Trained Fatigue	41.2 ± 4.7	61.0 ± 8.8
Non-Trained	63.7 ± 3.7	60.1 ± 3.8

TABLE 20

MEANS FOR PLASMA FFA
IN uEq/ml

Group	Diet	
	PW	AL
Trained Rest	0.272 $\pm$ 0.05	0.283 $\pm$ 0.01
Trained Fatigue	0.617 $\pm$ 0.04	0.619 $\pm$ 0.06
Non-Trained	0.290 $\pm$ 0.01	0.305 $\pm$ 0.01

TABLE 21

MEANS FOR RIGHT FAT PAD FFA
RELEASE IN uEq/gm.

Group	Diet	
	PW	AL
Trained Rest	1.97 ± 0.17	2.04 ± 0.23
Trained Fatigue	7.37 ± 0.46	6.62 ± 1.16
Non-Trained	2.12 ± 0.16	2.16 ± 1.00

TABLE 22

MEANS FOR LEFT FAT PAD FFA
RELEASE IN uEq/gm.

Group	Diet	
	PW	AL
Trained Rest	2.36 ± 0.36	2.00 ± 0.11
Trained Fatigue	6.69 ± 0.19	6.93 ± 0.20
Non-Trained	2.09 ± 0.12	2.08 ± 0.08

TABLE 23

MEANS FOR BLOOD GLUCOSE
IN MG%

Group	Diet	
	PW	AL
Trained Rest	151.0 ± 3.0	134.2 ± 10.4
Trained Fatigue	58.6 ± 7.8	79.8 ± 26.0
Non-Trained	136.5 ± 7.0	141.0 ± 4.0

APPENDIX C

SCHEFFÉ MULTIPLE COMPARISON TEST

<u>LEGEND FOR ALL TABLES IN APPENDIX C</u>

TR - TRAINED REST

TF - TRAINED FATIGUE

NT - NON TRAINED

TABLE 24

LIVER WEIGHT (gms)

Groups	TR	TF	NT
Means	15.1 (1)	13.4 (2)	15.7 (3)
1		1.9433	0.7971
2			3.2140**

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 25

HEART WEIGHT (mg)

Groups	TR	TF	NT
Means	1120.4 (1)	1182.5 (2)	1060.7 (3)
1		1.4821	1.6560
2			3.5534**

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 26

SPLEEN WEIGHT (mg)

Groups	TR	TF	NT
Means	656.0 (1)	569.2 (2)	702.2 (3)
1		1.5548	0.9644
2			2.8490*

t (0.05) > 2.5219*
t (0.01) > 3.1812**

TABLE 27

LEFT TESTICLE WEIGHT (mg)

Groups	TR	TF	NT
Means	1576.1 (1)	1331.4 (2)	1620.3 (3)
1		3.5353**	0.7648
2			4.9994**

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 28

RIGHT FAT PAD WEIGHT (mg)

Groups	TR	TF	NT
Means	1972.9 (1)	1714.4 (2)	2719.5 (3)
1		0.8169	2.7868*
2			3.8528**

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 29

LEFT FAT PAD WEIGHT (mg)

Groups	TR	TF	NT
Means	1952.5 (1)	1675.5 (2)	2743.5 (3)
1		0.8706	2.9241*
2			4.0534**

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 30

RIGHT ADRENAL WEIGHT (mg)

Groups	TR	TF	NT
Means	27.4 (1)	25.6 (2)	20.4 (3)
1		0.8531	3.8400**
2			2.9988*

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 31

LEFT ADRENAL WEIGHT (mg)

Groups	TR	TF	NT
Means	28.1 (1)	26.3 (2)	22.0 (3)
1		0.8256	3.2521**
2			2.4109

t(0.05) > 2.5179*
t(0.01) > 3.1654**

TABLE 32

LIVER GLYCOGEN (mg%)

Groups	TR	TF	NT
Means	41.4 (1)	3.4 (2)	31.4 (3)
1		7.9210**	2.5057
2			7.2589**

t (0.05) > 2.5219*
t (0.01) > 3.1812**

TABLE 33

GASTROCNEMIUS GLYCOGEN (mg%)

Groups	TR	TF	NT
Means	1.35 (1)	0.71 (2)	1.15 (3)
1		1.8812	0.6805
2			1.5736

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 34

BICEPS BRACHII GLYCOGEN (mg%)

Groups	TR	TF	NT
Means	2.02 (1)	0.99 (2)	1.79 (3)
1		2.3526	0.5698
2			2.2662

t (0.05) > 2.5357*
t (0.01) > 3.2047**

TABLE 35

PLASMA LACTATE (mg%)

Groups	TR	TF	NT
Means	31.6 (1)	172.7 (2)	36.8 (3)
1		16.5234**	0.7409
2			18.8161**

t (0.05) > 2.5219*
t (0.01) > 3.1812**

TABLE 36

GASTROCNEMIUS LACTATE (u MOLES/g)

Groups	TR	TF	NT
Means	55.6 (1)	47.8 (2)	61.6 (3)
1		1.2579	1.1119
2			2.7677*

t (0.05) > 2.5179*
t (0.01) > 3.1654**

TABLE 37

PLASMA FFA (uEq/ml)

Groups	TR	TF	NT
Means	.280 (1)	.618 (2)	.299 (3)
1		11.9014**	0.7600
2			14.3049**

t (0.05) > 2.5219*
t (0.01) > 3.1812**

TABLE 38

RIGHT FAT PAD FFA (uEq/gm)

Groups		TR	TF	NT
Means		2.02 (1)	7.05 (2)	2.14 (3)
	1		14.8948**	0.4916
	2			16.8439**

t (0.05) > 2.5338*
t (0.01) > 3.2000**

TABLE 39

LEFT FAT PAD FEA (uEq/gm)

Groups		TR	TF	NT
Means		2.12 (1)	6.78 (2)	2.09 (3)
	1		25.1484**	0.2173
	2			29.1304**

t (0.05) > 2.5318*
t (0.01) > 3.1969**

TABLE 40

BLOOD GLUCOSE (mg%)

Groups	TR	TF	NT
Means	139.0 (1)	65.1 (2)	139.3 (3)
1		6.5683**	0.0293
2			9.1018**

t (0.05) > 2.5417*
t (0.01) > 3.2187**

APPENDIX D

RAW DATA

TABLE 41

TOTAL BODY WEIGHTS OF PAIR-WEIGHED ANIMALS

TRAINED REST	
RAT #	BODY WEIGHT (GMS)
2	493
9	492
30	448
32	463
35	354

TRAINED FATIGUE	
RAT #	BODY WEIGHT (GMS)
1	416
5	391
6	484
12	444
25	389
28	410
36	489
56	448
80	458
83	461

TABLE 41 (Continued)

NON TRAINED	
RAT #	BODY WEIGHT (GMS)
13	480
14	491
15	419
18	450
19	484
21	478
39	477
43	355
44	414
46	488
47	379
48	408
67	479
94	474
96	412

TABLE 42

TOTAL BODY WEIGHTS OF AD LIB ANIMALS

TRAINED REST

RAT #	BODY WEIGHT (GMS)
97	403
98	343
99	442
100	408
101	455
105	472
106	434
108	376

TRAINED FATIGUE

RAT #	BODY WEIGHT (GMS)
31	381
84	419
102	529
103	301
107	447

TABLE 42 (Continued)

NON TRAINED	
RAT #	BODY WEIGHT (GMS)
27	396
41	483
50	571
71	449
72	472
95	525
109	450
110	466
111	416
112	487
113	484
114	456
115	484
116	414
117	552
118	393
119	517
120	475
121	468

TABLE 43

ORGAN AND MUSCLÉ WEIGHTS OF PAIR-WEIGHED
TRAINED ANIMALS SACRIFICED AT REST

Rat #	Liver gm	Heart mg	Right Kidney mg	Spleen mg	Right Adrenal mg	Left Adrenal mg	Right Testicle mg	Left Testicle mg	Right Fat Pad mg	Left Fat Pad mg	Right Gastrocnemius mg	Left Gastrocnemius mg	Left Biceps Brachii mg
2	15.3	1156.4	1640.0	719.1	26.5	25.6	1720.9	1711.9	2376.3	2680.5	1869.1	2208.2	270.0
9	17.6	1070.2	1518.2	612.2	17.2	16.8	1847.2	1772.0	2425.0	2095.8	2642.2	2681.8	270.0
30	16.8	1148.2	1428.2	571.8	29.6	27.4	1530.2	1475.2	1589.6	1877.8	2132.8	2088.6	263.8
32	18.0	1510.2	1519.0	745.2	25.8	22.2	1667.8	1683.4	2585.2	2457.2	1882.4	1667.8	250.4
35	11.1	871.0	1027.0	544.0	26.0	30.0	1548.0	1579.0	1410.0	1229.0	1413.0	1258.0	216.0

TABLE 44

ORGAN AND MUSCLE WEIGHTS OF PAIR-WEIGHED TRAINED ANIMALS SACRIFICED AT FATIGUE

Rat #	Liver gm	Heart mg	Right Kidney mg	Spleen mg	Right Adrenal mg	Left Adrenal mg	Right Testicle mg	Left Testicle mg	Right Fat Pad mg	Left Fat Pad mg	Right Gastrocnemius mg	Left Gastrocnemius mg	Left Biceps Brachii mg
1	12.4	1035.0	1255.0	500.0	28.0	24.0	1506.0	1429.0	2292.0	2418.0	1590.0	1701.0	217.0
5	11.2	1108.0	1314.0	492.0	22.0	24.0	1479.0	1479.0	1732.0	1620.0	1751.0	1727.0	183.0
6	15.8	1260.2	1486.0	550.4	30.4	31.6	1634.0	1634.0	2777.0	3047.0	2098.0	1994.0	288.0
12	18.4	1192.8	1550.0	601.0	30.0	32.9	1622.9	1586.4	2416.5	2383.5	2429.1	2314.6	290.0
25	10.4	1240.0	1360.0	422.0	24.0	24.0	1332.0	1213.0	1680.0	1608.0	2100.0	2550.0	184.0
28	11.8	1300.0	1247.0	1250.0	19.0	19.0	1804.0	1450.0	1113.0	1064.0	2045.0	2323.0	252.0
36	15.0	1314.2	1413.2	711.2	30.8	28.2	1577.6	1169.2	3225.2	3713.8	2452.4	2428.2	273.4
56	11.2	1223.2	1199.4	502.2	24.2	26.2	770.0	1001.6	308.2	174.2	2586.2	2351.4	261.4
80	15.0	1234.6	1463.4	433.0	29.8	33.8	-	-	-	-	3027.0	2379.2	261.8
83	13.0	1189.0	1456.4	570.0	23.2	22.1	1810.2	-	296.4	280.6	2129.6	1820.4	298.3

TABLE 45

ORGAN AND MUSCLE WEIGHTS OF PAIR-WEIGHED
NON TRAINED ANIMALS SACRIFICED AT REST

Rat #	Liver gm	Heart mg	Right Kidney mg	Spleen mg	Right Adrenal mg	Left Adrenal mg	Right Testicle mg	Left Testicle mg	Right Fat Pad mg	Left Fat Pad mg	Right Gastrocnemius mg	Left Gastrocnemius mg	Left Biceps Brachii mg
13	17.0	998.4	1285.0	788.2	25.6	23.6	1547.0	1497.6	3191.0	3060.2	2287.2	2308.6	252.8
14	15.6	1010.9	1463.8	670.9	18.9	24.4	1675.0	1641.0	3594.2	3666.5	2375.0	2330.5	272.9
15	14.0	1088.2	1250.2	720.4	10.2	13.8	1679.4	1640.6	2114.8	1901.6	1650.2	1981.4	236.4
18	13.8	1072.2	1241.8	675.6	22.2	18.6	1596.6	1613.6	4604.5	4024.4	2242.2	2145.8	202.4
19	17.2	1037.8	1422.6	697.0	24.0	23.8	1676.4	1770.4	3125.6	3291.4	2384.0	2618.8	264.6
21	14.6	1115.8	1423.8	759.8	12.0	15.6	1935.8	1948.2	4925.8	4703.0	2615.0	2422.8	285.0
39	16.6	1044.8	1335.4	701.6	13.2	13.2	1706.0	1644.0	1999.0	2196.4	2560.0	2287.8	312.0
43	11.8	758.0	1103.4	535.0	10.0	8.2	1650.2	1607.6	3500.0	4508.0	1388.0	1755.8	258.8
44	14.0	1095.0	1295.0	-	31.0	31.0	1556.0	1837.0	3096.0	2600.0	2231.0	-	228.0
46	17.4	1111.4	1371.5	668.7	19.8	19.8	1675.0	1549.5	2861.8	3059.4	2436.2	2700.2	248.4
47	14.2	958.0	1479.8	419.0	9.2	12.0	1357.4	1352.6	1620.8	1871.8	1888.2	1634.6	224.2
48	10.4	927.0	1411.0	1059.0	13.0	29.0	1726.0	1746.0	1721.0	1688.0	2225.0	1726.0	228.0
67	16.2	1099.2	1450.0	719.0	14.6	15.8	3106.6	1661.8	2923.8	2476.4	2090.8	1998.4	281.2
94	15.0	964.0	1417.4	583.7	21.8	17.7	1356.7	1362.3	3004.0	2823.6	2043.7	1962.6	250.4
96	14.8	965.0	1259.0	642.8	-	28.0	1735.6	1680.0	2687.5	2585.3	232.0	2189.6	220.0

TABLE 46

ORGAN AND MUSCLE WEIGHTS OF AD LIB TRAINED ANIMALS SACRIFICED AT REST

Rat #	Liver gm	Heart mg	Right Kidney mg	Spleen mg	Right Adrenal mg	Left Adrenal mg	Right Testicle mg	Left Testicle mg	Right Fat Pad mg	Left Fat Pad mg	Right Gastrocnemius mg	Left Gastrocnemius mg	Left Biceps Brachii mg
97	14.0	971.6	1278.2	650.0	28.3	24.7	1700.0	1650.0	1571.9	1631.0	1938.2	2008.2	223.8
98	13.2	1136.0	1589.0	761.0	29.0	31.0	1494.0	966.0	1270.0	1286.0	1518.0	1094.0	225.0
99	17.6	1164.2	1430.2	764.2	21.2	28.2	1347.4	1543.8	2333.2	2227.8	2109.2	2138.2	239.4
100	13.7	1040.3	1541.2	605.5	30.4	29.6	1583.9	1703.2	2350.2	2050.9	2078.6	2028.2	253.2
101	16.4	1064.6	1449.2	807.3	31.0	33.7	1776.6	1782.8	2390.1	2724.2	2303.1	2204.6	255.5
105	16.4	1288.4	1442.0	668.4	32.0	33.4	1643.6	1674.0	1929.6	1679.2	2427.6	2353.0	292.4
106	16.0	1104.0	1255.6	711.9	29.1	32.8	1551.9	1590.1	1820.0	1847.3	2176.4	1954.2	236.5
108	10.4	1040.2	1306.2	367.8	29.8	29.8	1380.4	1358.2	1596.2	1596.2	1719.6	1820.6	200.0

TABLE 47

ORGAN AND MUSCLE WEIGHTS OF AD LIB TRAINED ANIMALS SACRIFICED AT FATIGUE

Rat #	Liver gm	Heart mg	Right Kidney mg	Spleen mg	Right Adrenal mg	Left Adrenal mg	Right Testicle mg	Left Testicle mg	Right Fat Pad mg	Left Fat Pad mg	Right Gastrocnemius mg	Left Gastrocnemius mg	Left Biceps Brachii mg
31	12.6	1064.0	1299.0	480.0	18.0	18.0	1412.0	1403.0	1384.0	1201.0	1719.0	1677.0	226.0
84	11.6	1084.3	1176.6	433.8	28.2	33.6	1054.6	1116.4	1426.7	1671.4	2334.6	2384.6	251.9
102	15.4	1300.2	1572.6	473.0	16.2	14.2	1530.0	1507.2	2773.5	2671.0	2227.2	2194.4	316.6
103	11.1	1044.0	1350.0	-	28.0	31.0	1376.0	1412.0	615.0	564.0	741.0	967.0	211.0
107	16.0	1147.6	1570.2	549.8	32.4	32.0	1868.0	907.8	1962.4	1040.0	2124.4	2201.4	301.8

TABLE 48

ORGAN AND MUSCLE WEIGHTS OF AD LIB NON TRAINED ANIMALS SACRIFICED AT REST

Rat #	Liver gm	Heart mg	Right Kidney mg	Spleen mg	Right Adrenal mg	Left Adrenal mg	Right Testicle mg	Left Testicle mg	Right Fat Pad mg	Left Fat Pad mg	Right Gastrocnemius mg	Left Gastrocnemius mg	Left Biceps Brachii mg
27	14.2	942.8	1217.2	599.2	25.0	26.2	1400.2	1560.0	1697.8	2103.2	2006.2	1080.4	240.2
41	16.8	1162.2	1553.9	749.4	20.2	20.4	1570.4	1553.8	1599.8	2593.2	2525.2	2480.8	270.2
50	21.2	1224.6	1522.0	816.4	27.3	28.2	1849.8	1849.8	4625.9	4611.8	2687.8	2067.8	213.0
71	13.8	973.2	1216.2	694.2	19.4	22.2	1590.8	1471.8	1991.6	1772.2	2123.2	1774.2	323.8
72	17.2	1017.8	1282.2	792.8	21.4	23.8	1532.0	1532.6	1952.4	2181.8	2424.8	2489.4	323.8
95	19.2	1165.6	1373.6	792.4	26.0	25.4	1505.6	1568.8	3118.9	3023.3	1515.8	1058.2	342.3
109	16.2	1148.0	1249.0	-	24.0	28.0	1596.0	1617.0	1909.0	1864.0	2220.0	1964.0	257.0
110	18.4	1092.0	1269.0	-	20.0	23.0	1598.0	1576.0	2975.0	3032.0	1923.0	2168.0	282.0
111	15.2	925.6	1250.2	565.8	9.0	9.6			1825.4	2139.0	2023.4	1967.0	227.8
112	19.2	1225.8	1603.8	792.6	27.2	26.4	1061.0	1677.8	1792.4	2105.8	2234.0	2345.4	260.4
113	15.6	1063.7	1320.9	681.9	20.9	22.8	1661.9	1684.6	2040.0	2054.6	1860.9	2264.9	271.9
114	16.4	1252.0	1269.4	625.6	12.0	11.8	1581.6	1598.8	2550.2	2547.0	2024.2	2143.0	248.6
115	14.6	1160.2	1466.8	873.0	28.2	28.2	1880.0	1679.2	2757.5	2674.5	2425.2	2221.2	254.0
116	12.0	1022.0	1234.6	630.2	22.4	21.8	1634.6	1590.6	2672.6	2701.0	1642.2	1723.4	180.2
117	18.4	1134.0	1440.6	655.5	28.4	26.0	1680.0	1720.0	2773.0	2711.4	2853.0	2832.4	330.0
118	13.0	1005.4	1215.4	692.2	17.8	25.8	1718.6	1680.2	1960.2	2572.2	2107.6	2143.4	230.2
119	17.0	1168.2	1427.8	742.2	22.2	27.8	1488.2	1457.8	3487.6	3431.0	2306.3	2878.8	296.2
120	16.7	1086.0	1466.0	798.6	25.4	24.3	1713.8	1647.6	2232.7	1962.8	2376.3	2070.6	234.6
121	17.2	1047.2	1394.2	625.2	31.8	30.0	1508.0	1450.4	3532.8		1544.0	1776.4	272.4

TABLE 49

PAIR-WEIGHED TRAINED ANIMALS
SACRIFICED AT REST

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
2	30.6	0.51	2.75	26				3.12	148
9	58.3	0.82	1.17	35	41	.361	2.11	2.52	154
30	47.3	0.68	0.89	34	71	.235	-	2.41	
32	46.2	0.72	0.97	37	71	.221	2.16	1.38	
35	25.1	2.34	3.01	18	64		1.63		

TABLE 50

PAIR-WEIGHED TRAINED ANIMALS
SACRIFICED AT FATIGUE

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
1	2.84	0.38	0.64	200	31	.863	7.94	6.47	51
5	4.10	1.06	1.13	100	50	.639	6.78	6.37	72
6		0.61	0.83	100	52	.499	8.34	7.41	29
12	7.64	0.63	0.70	-	13	.684	6.42	6.78	-
25	1.53	0.51	0.67	220	59	.581		6.43	107
28	8.16	0.64	0.51	132	48	.623			29
36	1.62	0.53	0.77	170	30	.540			54

TABLE 50 (continued)

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
56	1.78	0.55	0.77	170	28	.459			58
80	2.65	0.46	0.61	200	44	.537			60
83	0.64	0.55	0.83	200	57	.741			67

TABLE 51

PAIR-WEIGHED NON TRAINED ANIMALS
SACRIFICED AT REST

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
13	58.6	0.63		42	72	.263			131
14	21.7	0.91	1.55	21	31	.300	2.12	2.13	128
15	29.3	1.03	2.41	42	92	.321	2.33	2.31	116
18	38.5	1.67	2.04	37	69	.381	3.64	1.37	93
19	18.7	0.92	1.17	18		.318	1.94	1.76	157
21	40.3	0.80		44	55	.201	2.02	2.41	
39	47.7	0.91		44	51	.198	1.78	1.64	174
43	23.4	1.01	1.15	63	65	.291	3.11	1.83	142

TABLE 51 (continued)

RAT #	GLYCOGEN Liver mg %	GLYCOGEN Gastroc mg %	GLYCOGEN Bicep mg %	LACTATE Plasma mg %	LACTATE Gastroc u moles/gm	FREE FATTY ACIDS Plasma uEq/ml	FREE FATTY ACIDS Right Fat Pad uEq/gm	FREE FATTY ACIDS Left Fat Pad uEq/gm	GLUCOSE Plasma mg %
44	23.3	-	4.42	48	60	.298	1.56	1.69	147
46	33.7	0.59	-	45	61	.341	1.97	2.31	
47	29.4	1.49	1.29	-	78	-	2.31	2.02	-
48	18.2	2.14	4.08	-	69	-	2.08	1.96	-
67	35.7	0.84		24	62	.257	1.78	2.13	140
94	38.6	0.68		31	57	.304	1.63	3.02	137
96	32.4	1.17	1.05	45	70	.300	1.34	2.64	

TABLE 52

AD LIB TRAINED ANIMALS SACRIFICED
AT REST

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
97	43.7	0.84	2.65	25	35	.241	1.83	2.41	123
98	31.1	2.59	3.57	24	62	.300	1.23	1.61	
99	43.8	0.79	1.07	40	64	.304	2.14	1.79	
100	26.0	0.74	0.90	35	29	.322	3.31	1.78	
101	37.7	5.26	3.23	37	49	.205	1.92	1.86	
105		0.78		33	37	.300	2.13	1.93	147
106	65.1	1.03		35	69	.312	1.32	2.22	124
108		0.50		32	77	.280	2.44	2.43	109

TABLE 53

AD LIB TRAINED ANIMALS
SACRIFICED AT FATIGUE

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
31		1.39	1.75		72	.583			113
84	2.28	0.53	0.77	200	74	.631	4.41	6.67	31
102	5.16	0.59	0.75		49	.592	7.13	6.78	
103		1.72	3.33	200	78	.478			135
107	1.97	0.53	0.82	180	32	.813	8.31	7.33	40

TABLE 54 (Continued)

AD LIB NON TRAINED ANIMALS
SACRIFICED AT REST

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
27	32.6	0.702		32	76	.319	1.63	1.14	100
41	35.8	0.67	0.94	40	69	.302	2.33	2.31	144
50	21.3	0.29		32	27	.341	1.98	2.07	
71	27.1	0.58		31	43	.263	2.31	2.62	
72	50.8	1.64	2.75	26	79	.242	1.37	2.43	148
95	18.7	2.17	1.01	34	72	.394	3.32		153
109	16.6	4.24	4.00	39	74	.316	2.31	2.16	153
110	21.1	3.49	4.36	30	84	.298	2.43	1.76	132

TABLE 54 (continued)

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
111	43.2	0.76	0.92	46	56	.300	2.41	2.33	
112	43.7	0.74	0.83	90	70	.253	2.41	2.30	153
113	18.3	0.89		25	34	.317	2.13	2.32	137
114	60.9	0.71	1.05	31	59	.312	2.14	2.12	158
115	12.9	1.02	0.98	21	66	.316	2.32	1.91	142
116	18.6	1.51	0.71	39	68.	.302	1.82	2.16	121
117	44.4	1.02	1.15	30	48	.330			131
118	19.5	0.68	0.85	31	41	.300	1.87	1.96	142

TABLE 54 (continued)

RAT #	GLYCOGEN			LACTATE		FREE FATTY ACIDS			GLUCOSE
	Liver mg %	Gastroc mg %	Bicep mg %	Plasma mg %	Gastroc u moles/gm	Plasma uEq/ml	Right Fat Pad uEq/gm	Left Fat Pad uEq/gm	Plasma mg %
119	26.2	0.44	0.86	53	63	.278	1.87	1.93	163
120	40.9	0.62	0.87	25	41	.264	1.97	1.87	153
121	26.9	0.94	2.47	20	72	.341	2.31	2.04	126